

Diseases of the Nose, Throat, and Ear

INCLUDING BRONCHOSCOPY AND ESOPHAGOSCOPY

Edited by

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With the Collaboration of

SIXTY-FOUR OUTSTANDING AUTHORITIES

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PREFACE

In the preparation of this textbook it has been our purpose to present the various phases of modern otorhinolaryngology and bronchoesophagology in a form most practical for those interested in these specialties, be they students, teachers, specialists, or general practitioners. In order that the book should be authoritative, contributors have been chosen from among those who are not only thoroughly acquainted with the literature of their respective subjects but who have had extensive clinical and teaching experience as well as active part in the epoch-making developments of recent years.

Text matter and illustrations have been planned with the thought in mind of rendering accepted facts and opinions of today quickly accessible. Except for a few classics, new illustrations have been specially prepared, and, as far as the respective subjects would permit, labels, rather than letters or figures referring to explanations in the appended legends, have been placed directly on the drawings. For lucidity, eighteen of the illustrations have been selected for full color reproduction.

In the text the time proven plan of organization has been followed. The anatomy and physiology of the various organs have been discussed, followed by a description of the method of examination. The diseases, disorders, malformations, anomalies, and other conditions, such as those due to trauma or the presence of foreign bodies, have been covered under the

headings, wherever appropriate, of "etiology," "pathology," "symptoms," "diagnosis," "complications," "treatment," "sequelae," and "prognosis," the emphasis being placed throughout on what to do and how to do it. On controversial subjects, overlapping of text matter has been utilized to present fairly two or more contributors' viewpoints. Procedures that have not stood the test of clinical experience have been omitted, except when mention is needed to indicate pitfalls, recent therapeutic adjuncts, however, whether of proven worth or of promise, have been given emphasis. The articles on endoscopic photography, aviation otolaryngology, and chemotherapy in otolaryngology will, we believe, be of particular general interest.

No attempt has been made to give any historical notes. The history of medicine is intensely absorbing, but it calls for an entirely different type of book. The reader interested in historical matters, or in literary research, will, doubtless, be interested in consulting the list of references following each article.

Our thanks are due to each and every contributor for invaluable cooperation, and to the personnel of W. B. Saunders Company for unfailing courtesy and kind helpfulness.

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PART I. NOSE AND NASAL ACCESSORY SINUSES

ANATOMY OF THE NOSE AND PARANASAL SINUSES

THE NOSE

For purposes of description the nose is usually divided into an external nose and an internal nose.

The External Nose—The external nose or outer nose is familiar to all in its many variations in size and shape. The more or less pointed tip is known as the *apex*. The aspect directed caudally or inferiorly and between the apex and the upper lip is the *base*, in which are the two openings into the nasal cavities called the *anterior nares*. Where the nose merges with the forehead is the *root* of the nose and extending from the root to the apex is the *dorsum* of the nose, the portion of this near the root being the *bridge*. The rounded eminence to the side of each naris is called the wing of the nose or the *ala nasi*.

Framework of the External Nose—The framework of the external nose is partially bony and partially made up of cartilages of the hyaline type. The aperture of the bony part of the external nose is somewhat the shape of an inverted, blunt pointed valentine heart, the lower two thirds of this aperture being bounded by the maxillae and the upper third by the two nasal bones which are interposed between the frontal processes of the two maxillary bones. The nasal bones (Fig. 1) form the framework of the bridge of the nose and are of course, subject to variation which ranges to occasional complete absence.

The cartilaginous portion of the framework of the external nose consists of five major cartilages—the right and left *greater alar cartilages*, right and left *lateral nasal cartilages* and the margin of the unpaired *septal cartilage* which to a varying extent is interposed between the lateral nasal and greater alar cartilages of the right and left sides along the dorsum of the nose—and of several smaller cartilaginous bits some called *lesser alar cartilages*, which are posterior to the greater alar cartilage and supe-

rior to the fibrofatty tissue of the ala nasi and others called *sesamoid cartilages* which may be present between the greater alar cartilage and the lateral nasal cartilage. The relationship which these cartilages have to each other as they contribute to the framework of the external nose is shown in Figures 1 and 2. The five major cartilages may be variously fused to one another and have been interpreted by some as being all one piece.

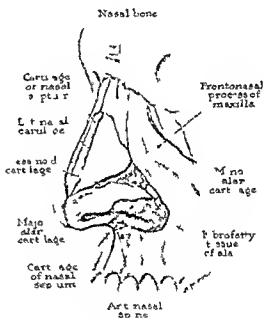


Fig. 1—The framework of the external nose

Muscles of the External Nose—The musculature of the nose varies a great deal in its degree of development and is described differently by various authors. The right and left *procerus muscles* are usually combined into one muscle running from the lower part of the nasal bones and adjacent parts of the lateral cartilages to the skin at the root of the nose where it blends with the medial fasciculi of the frontalis muscles thereby being able to produce horizontal wrinkling of the skin on the bridge of the nose. Arising from the maxilla near the apices of the incisor and canine teeth are the *depressor septi* (medially) and the *nasalis* (laterally) the former

inserting into the mobile part of the septum and the posterior part of the margin of the naris, and the latter inserting partly into the ala nasi and partly continuing superiorly to expand into an aponeurosis which joins the one of the opposite side on the dorsum of the nose. The name "depressor septi" indicates the action of this muscle. Most of the nasalis apparently tends to depress the ala and press it medially. However, probably some fasciculi, which run from the aponeurosis over the dorsum of the nose downward to end in the ala, together with some fasciculi of the angular head of the quadratus labii superioris which also end in the ala, tend to elevate the ala. There is *dilatator musculature* described which is thin and indefinite.

Skin of the Nose—Over the bony portion of the external nose the skin is thin and since it rests upon a loose subcutaneous tissue contain-

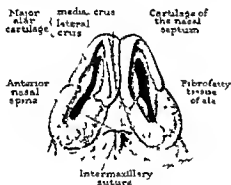


Fig 2—The framework of the external nose as seen from below

ing very little adipose it is freely movable. As the tip and base of the nose are approached, the skin becomes a little thicker and the subcutaneous tissue is much denser, becoming fibrofatty in nature, which means that the skin loses its movability. It also acquires many more large sebaceous glands, the openings of which in relation to the very fine hairs are readily seen with the unaided eye.

Blood Supply of the External Nose—Probably to compensate for its exposed position, the arterial network supplying the external nose is a rich one, which is contributed to from several sources. Coming onto the nose near its root, is the *dorsal nasal* branch of the ophthalmic branch of the internal carotid artery. From the side comes a small branch of the infra orbital of the internal maxillary branch of the external carotid. The *lateral nasal* branch of the external max-

illary branch of the external carotid comes onto the nose just above the ala and the *angular* branch of the external maxillary runs superiorly at the side of the nose to anastomose with the dorsal nasal artery. A small *external nasal* branch comes through onto the dorsum just below the nasal bone from the anterior ethmoidal artery (p. 8). From below, the nose receives an *alar* and a *septal* branch from the superior labial branch of the external maxillary artery.

There is some venous drainage accompanying all of the arteries just described but the chief drainage is by way of the *anterior facial vein* which has tributaries corresponding to the branches of the external maxillary artery. The anastomosis of the angular vein with the ophthalmic vein may become of significance since the ophthalmic vein empties into the cavernous sinus.

Lymphatics of the External Nose—There are rich lymphatic capillary networks in the skin, muscles, periosteum, and perichondrium of the external nose, particularly dense at the apex, alae, and root. The principal drainage from these networks is by way of vessels which follow the anterior facial vein to the submaxillary group of nodes. Additional drainage from the region of the root and side of the nose has been described as taking place by way of vessels passing laterally through the upper and lower eyelids to get to the parotid nodes, but this drainage is denied (Rouviere). There is anastomosis with the lymphatics of the mucous membrane of the nasal fossae.

Nerve Supply of the External Nose—The muscles of the external nose are supplied by branches from the buccal branches of the facial or seventh cranial nerve. The skin is supplied by the trigeminal or fifth cranial nerve through the *infratrochlear* (region of the root) and *external nasal* (onto the dorsum just below the nasal bone from within the nose) branches of the nasociliary branch of the ophthalmic division and through the nasal branches (much of side of nose, ala, and region of anterior naris) of the infra-orbital branch of the maxillary division.

The Internal Nose.—The internal nose comprises the cavity of the nose and, of course, the walls of the cavity. The general cavity is divided by a septum into a right and a left *nasal fossa* (nasal chamber or nasal cavity). Each fossa is usually described as having a floor, a roof, a medial wall (nasal septum), and a lateral wall

and it opens by way of an *anterior naris* or nostril to the exterior and by way of a *posterior naris* or choana into the nasopharynx. The shape of the fossa as seen in a coronal section is roughly that of a rather long and narrow right angle triangle, with the right angle at the junction of the medial wall and floor and with the superior angle blunted by the roof of the fossa. The general shape of the fossa as seen in a sagittal section is obvious in Figure 3.

Anterior Naris and Vestibule—The anterior nares are in the base of the nose and therefore look caudalward. They present many individual

skin which is tightly adherent to the underlying cartilage and fibrofatty tissue and contains, in the lower part of the vestibule, coarse hairs known as *vibrissae* which curve downward to guard the entrance. There are sebaceous glands related to these hairs.

Posterior Naris—Each posterior naris or choana is a roughly oval opening approximately an inch in its vertical diameter and a half inch in its transverse diameter in the adult. It is completely bounded by bone covered by mucoperiosteum. Above is the body of the sphenoid and ala of the vomer, medially, the

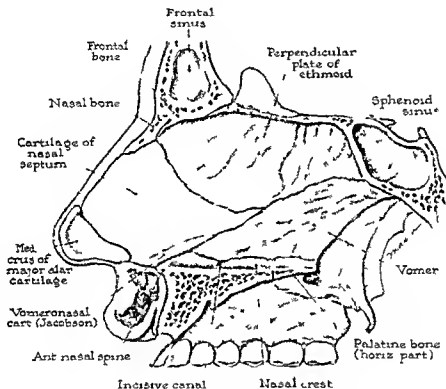


Fig. 3—The framework of the septum of the nose

and racial variations in size, shape, and the exact plane which they occupy. Directly above the naris is the slightly expanded part of the nasal cavity which is called the vestibule. The naris and vestibule are bounded by the medial and lateral crura of the greater alar cartilage and the fibrofatty tissue of the ala (Fig. 2). Corresponding to the lower border of the lateral cartilage of the nose is a slight elevation on the lateral wall of the nasal cavity called the *lumen* which forms a superior limit of the vestibule. A *recess of the vestibule* extends into the apex of the nose anterior to the anterior margin of the anterior naris. The vestibule is lined by a thin

posterior free margin of the vomer, laterally, the medial pterygoid plate of the sphenoid bone and below, the posterior border of the horizontal part of the palatine bone.

The posterior nares are remarkably equal in size as the septum does not deviate in this region. There are, however, occasional instances of congenital atresia.

Roof of the Nasal Fossa—The roof, as usually considered, is in the form of an arch extending from the tip of the nose to the superior limit of the posterior naris. It is little more than a groove for most of its extent but does widen to 4 or 5 mm. in its posterior part. The struc-

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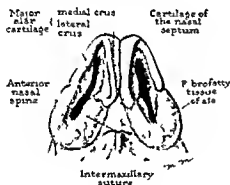


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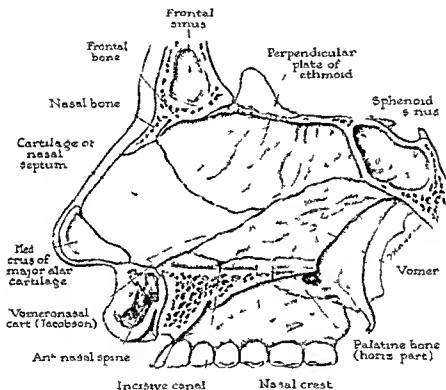


Fig. 3.—The framework of the septum of the nose

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tures forming the framework of the roof are shown in Figure 3. Some authors have named only that portion formed by the cribriform plate of the ethmoid as the roof, with the other parts as anterior and posterior walls of the fossa respectively.

Floor of the Nasal Fossa.—The floor of the nasal fossa has a bony framework formed for about its anterior three-quarters by the palatine process of the maxilla and for its posterior quarter by the horizontal process of the palatine bone. It occupies approximately a horizontal plane but it may slant downward in its posterior portion and frequently there is an elevation of the anterior margin of the bony floor. Laterally and medially the floor curves upward as it meets the lateral and medial walls of the fossa. The bony framework diminishes in thickness from the anterior to the posterior portion.

There is a bony canal transmitting anastomotic connections between blood vessels of the nasal and oral cavities which begins very close to the septum a centimeter or so from the anterior limit of the floor and runs downward and forward to end in common with the one of the other side in the incisive foramen. In the early development of the individual there is a canal of mucous membrane (the incisive duct) connecting the nasal and oral cavities but this usually becomes obliterated early leaving only a cord of cells and at times a depression in the mucous membrane at the nasal end of the canal the *nasopalatine recess*. A smaller bony canal usually in common with the one of the other side and posteromedial to the one just described transmits a branch of the nasopalatine nerve.

It probably should be pointed out that the width of the floor of the fossa is to an extent dependent on the size of the maxillary sinus.

Medial Wall of the Nasal Fossa or Nasal Septum.—The three main constituents of the framework of the nasal septum—the *vomer*, the *perpendicular plate of the ethmoid bone* and the *septal cartilage*—are related to each other, in general as shown in Figure 3. Contributing slightly to the margin of the septal framework are the anterior nasal spines of the maxillae, the nasal crest of the maxillary and palatine bones, the rostrum and crest of the sphenoid bone, the nasal spine of the frontal bone and the crests of the nasal bones all of which are for the articulation of the three main constituents of the septum named above. In relation to the caudal border of the septal cartilage on each

side of it there is a narrow strip of cartilage, the *vomeronasal cartilage* which in man has no close relation to the rudimentary organ of that name to be described presently. Beyond the antero-inferior margin of the septal cartilage, the medial crura of the greater alar cartilages (Figs. 1, 2) are the chief framework of this small portion of the septum and they together with the adjacent soft tissue can be pulled downward and wiggled around hence the name *mobile part of the septum* is frequently applied.

In the mucous membrane of the septum not far from the floor and a centimeter or a little more posterior to the posterior margin of the anterior naris there is a minute opening into a blind pouch 2 to 9 mm. in length running backward from the opening. This is the rudimentary *vomeronasal organ of Jacobson*. There is usually a thickening of the mucous membrane of the septum opposite the anterior end of the middle concha called the *tubercle of the septum* and early in life there are 4 to 6 obliquely running ridges on the postero-inferior part of the septum called the *folds of the septum*.

In the majority of older children and adults there is some degree of asymmetry of the septum either in the form of a deflection or the presence of ridges or spurs. Since this asymmetry is not as a rule present in the infant, it may be due either to trauma perhaps slight or to developmental factors possibly such as buckling due to overgrowth.

Lateral Wall of the Nasal Fossa.—As previously described (p. 3) not far above the anterior naris there is an elevation on the lateral wall of the nasal fossa called the *limen* which indicates the boundary between the vestibule and the rest of the nasal fossa or what is frequently referred to as the *nasal fossa proper*. The lateral wall of the nasal fossa proper presents several elevations or projections (Fig. 4) all with bony foundations which are thought of as subdividing the nasal fossa proper for purposes of description. Four of them running more or less horizontally and diminishing in length from the lowest to the highest are named the *inferior*, *middle*, *superior*, and *supreme nasal concha* or *turbinate* respectively. The portion of the nasal fossa overlapped by each concha is called a *meatus* of corresponding designation. The part of the fossa above the supreme concha is called the *spheno-ethmoidal recess*, the part between the conchae and the septum the *com-*

mon nasal meatus and that part posterior to the posterior ends of the conchae the *naso pharyngeal meatus*. More or less parallel to the dorsum of the nose and about halfway between it and the anterior end of the middle concha is a ridge known as the *aggr nasi* which separates the *olfactory sulcus* anterosuperior to it from the *atrium of the middle meatus* posterior to it.

The conchae meatuses and sphenoidal recess require some further description

and the opening being quite variable in different individuals. If it is close to the attachment of the concha it is usually a definite hole which remains open. If it is several millimeters below the attachment of the concha it is usually slit like and often guarded by a fold of mucous membrane called the valve of Hasner.

MIDDLE CONCHA—The bony framework of the middle concha is a projection of the ethmoid bone. The mucous membrane particularly at

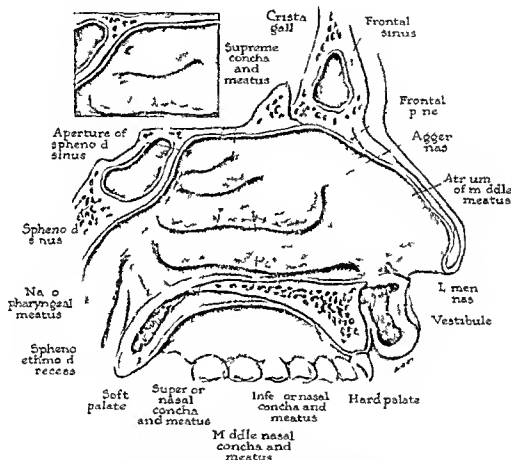


Fig. 4—The lateral wall of the left nasal fossa. The inset shows the posterosuperior part of the wall when the supraorbital concha is not present.

INFERIOR CONCHA—The bony framework of the inferior concha is a separate bone which articulates with the lacrimal bone, the conchal crest of the maxilla, the uncinate process of the ethmoid bone, and the conchal crest of the palatine bone. The mucous membrane of the inferior concha is thick and contains numerous venous plexuses forming a cavernous tissue of an erectile nature.

INFERIOR MEATUS—The nasolacrimal duct opens into the inferior meatus near its anterior

margin and posterior extremity is similar to that of the inferior concha.

MIDDLE MEATUS—When the middle concha has been removed (Fig. 6) a rather deep crescentic groove, the *infundibulum* or ethmoid infundibulum, is seen in the lateral wall of the middle meatus with a rounded elevation, the *ethmoid bulbo*, posterosuperior to it. The opening into the groove which is bounded antero-inferiorly by the mucous membrane covering the uncinate process of the ethmoid bone is called the *hiatus*

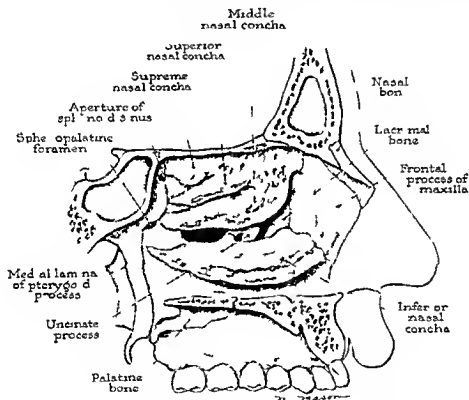
semilunaris The infundibulum ends antero superiorly in one of two ways. It is either continuous with the frontonasal duct of the frontal sinus or probably a little more commonly it continues into an anterior ethmoid air cell in which case the frontal sinus opens directly into a frontal recess of the middle meatus. The infundibulum has openings of other anterior ethmoid cells near its anterosuperior end and somewhere near or posterior to its middle is the main opening of the maxillary sinus. There may be accessory openings of the maxillary sinus either farther back in the infundibulum or in

more openings of various size of the posterior ethmoid air cells.

SUPREME CONCHA—In the neighborhood of 60 per cent (Schaeffer) of the instances that which in the other 40 per cent is all superior concha is split into two parts the upper of which is called the supreme or highest concha.

SUPREME MEATUS—The supreme meatus is very small but in 75 per cent (Schaeffer) has an opening of a posterior ethmoid air cell.

SPHENO-ETHMOIDAL RECESS—The sphenoidal recess is between the supreme concha or if that is not present the superior



5—The framework of the lateral wall of the left nasal fossa

the lateral wall
infundibulum
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concha and the roof of the nasal fossa. The ostium of the sphenoid sinus is on its posterior wall.

Mucous Membrane of the Nasal Fossa—In the general region of the lining there is a mucous membrane from the modified skin lining the nasal fossa to the mucous membrane lining the nasal cavity proper of which there are two parts: the *factory mucous membrane* and the *respiratory mucous membrane*. One general feature of the mucous membrane is its firm attachment to the underlying periosteum or forming a mucoperiosteum or

mucoperichondrium, as the case may be, which cannot be readily divided into its two parts but can quite readily be lifted off from the underlying bone or cartilage

The exact distribution of the olfactory mucous membrane is not agreed on by all and the area, in all probability, has rather irregular margins but, in general, it can be said to be on the medial surface of the superior concha and the adjacent part of the septum with overflow onto the anterosuperior part of the middle concha and the anterior part of the supreme concha. It is described as having a yellowish

plate of the ethmoid bone to end in the olfactory bulb. The tunica propria of the olfactory mucous membrane is made up of fibro elastic tissue and it contains tubulo-alveolar glands, the *olfactory glands (of Bowman)*, which resemble serous glands but may have a specific secretion in some way related to the reception of stimuli by the receptors of the olfactory cells

The respiratory mucous membrane which lines that part of the nasal fossa proper not lined by olfactory mucous membrane is also made up of an epithelium and a tunica propria

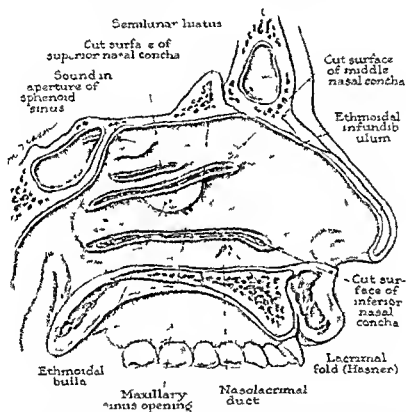


Fig 6—The lateral wall of the left nasal fossa with the middle and inferior conchae removed

tinge. The epithelium of the olfactory mucous membrane is a stratified epithelium with many so called supporting cells among which are placed the *olfactory cells* which receive the stimuli giving rise to the sense of smell. The olfactory cells have a fusiform cell body near the middle of the epithelial layer with a peripheral and a central process. The peripheral process extends to the surface of the epithelium, projecting a little, with several cilia like structures on its end. The central processes of the olfactory cells bundle together to form the olfactory nerves which pass through the cribriform

The epithelium is, for the most part, of the ciliated pseudostratified columnar variety with many goblet cells, although it may vary from this in some areas. The fibro elastic tissue of the tunica propria is looser near the epithelium, rich in cells, and has collections of lymphocytes. The tunica propria also contains numerous tubulo alveolar glands which are mucous in type with serous crescents. In the tunica propria especially on the inferior concha, the inferior margin and posterior border of the middle concha and, to some extent, on the related part of the septum, there are rich venous plexuses

semilunaris The infundibulum ends antero superiorly in one of two ways. It is either continuous with the frontonasal duct of the frontal sinus or, probably a little more commonly, it continues into an anterior ethmoid air cell, in which case the frontal sinus opens directly into a frontal recess of the middle meatus. The infundibulum has openings of other anterior ethmoid cells near its anterosuperior end and somewhere near or posterior to its middle is the main opening of the maxillary sinus. There may be accessory openings of the maxillary sinus either farther back in the infundibulum or in

more openings of various size of the posterior ethmoid air cells.

SUPREME CONCHA—In the neighborhood of 60 per cent (Schaeffer) of the instances, that which in the other 40 per cent is all superior concha is split into two parts, the upper of which is called the supreme or highest concha.

SUPREME MEATUS—The supreme meatus is very small but in 75 per cent (Schaeffer) has an opening of a posterior ethmoid air cell.

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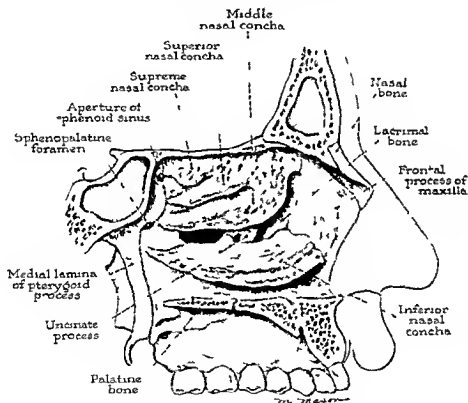


Fig 5—The framework of the lateral wall of the left nasal fossa

the lateral wall of the middle meatus below the infundibulum. The ethmoid cells which form the bulla ethmoidalis—called anterior by some, middle by others—usually open into the *suprabullar recess*.

SUPERIOR CONCHA—The bony framework of the superior concha is a projection of the ethmoid bone. The mucous membrane is much thinner than that of the middle and inferior conchae.

SUPERIOR MEATUS—The superior meatus is only about half the length of the middle meatus. In its upper and front part are one or

concha and the roof of the nasal fossa. The ostium of the sphenoid sinus is on its posterior wall.

Mucous Membrane of the Nasal Fossa—In the general region of the limen, there is a transition from the modified skin lining the vestibule (p. 3) to the mucous membrane lining the nasal fossa proper, of which there are two kinds, *olfactory mucous membrane* and *respiratory mucous membrane*. One general feature of the nasal mucous membrane is its firm blending with the underlying periosteum or *perichondrium* forming a *mucoperiosteum* or

pterygopalatine canal, (4) the septal branch of the superior labial branch of the external maxillary, (5) branches from the lateral nasal branch of the external maxillary to the lateral wall near the anterior naris, (6) branches which may perforate from any of the arteries supplying the external nose.

The blood from the rich venous plexus in the lining of the nasal fossa, which, as has been pointed out (p 8), forms a cavernous tissue especially on the inferior concha, parts of the middle concha, and lower part of the septum, is picked up by veins which more or less follow all of the arterial channels described above. Similar to the arterial arrangement, the largest of the veins is the *sphenopalatine vein* which empties into the pterygoid plexus of veins which in turn becomes the internal maxillary vein.

The anastomoses between the ethmoidal veins on the one hand and the meningeal veins and the superior sagittal sinus on the other hand should be mentioned because of their possible clinical importance.

Lymphatic Drainage of the Walls of the Nasal Fossa—There is a fairly well developed lymphatic capillary network throughout the lining of the nasal fossa, varying, of course, in its degree of development in different areas. The network in the olfactory area is said to be more or less separated from that in the respiratory area. The vessels collecting lymph from about the posterior two thirds to three quarters of the lymphatic capillary network run posteriorly, many of them forming a plexus just in front of the pharyngeal ostium of the auditory tube, and eventually drain into the retropharyngeal and superior deep cervical groups of nodes. The vessels collecting lymph from the anterior part of the network (in the walls of the vestibule and adjacent part of the fossa proper) communicate with the vessels of the external nose either at the region of the anterior naris or by piercing the framework of the wall of the nose, and drain eventually to the submaxillary group of nodes. There is some communication between the lymphatics of the right and left sides particularly in the region of the mobile septum.

There is some sort of connection between the cranial subarachnoid space and the lymphatics of the walls of the nasal fossa but there is no agreement as to whether or not this is a direct connection, or as to whether or not the perineural continuations of the subarachnoid space along the olfactory nerves are involved. It is

probable, however, that there are small special canaliculi leading directly from the subarachnoid space through the cribriform plate of the ethmoid bone to the lymphatic capillaries (Rouviere).

Nerve Supply of the Walls of the Nasal Fossa—As has been previously described (p 5), the central processes or axones of the olfactory cells, whose cell bodies are in the olfactory portion of the nasal mucous membrane, collect into bundles of nerve fibers called the *olfactory nerves* which converge toward the cribriform plate of the ethmoid bone (Fig 8) and pass through the small holes in this bony structure in order to enter the olfactory bulb which is in the anterior cranial fossa just above the cribriform plate. The above neurones are the first order neurones of the pathways involved in the sense of smell.

The innervation of the walls of the nasal fossa, in addition to that just described, is taken care of by branches of the ophthalmic and maxillary divisions of the trigeminal or fifth cranial nerve (Fig 8). From the ophthalmic division is the terminal portion of the *nasociliary nerve*, called by some the "anterior ethmoidal nerve," which enters the nasal mucous membrane with the anterior ethmoidal artery (p 8) near the anterior end of the cribriform plate of the ethmoid bone, and divides into *medial* and *lateral internal nasal* branches. The medial internal nasal branch spreads out on the anterosuperior part of the septum while the lateral internal nasal distributes to a similar part of the lateral wall and sends an external nasal branch through to the skin of the dorsum of the nose (p 2).

The *posterior superior nasal nerves* which are branches of the sphenopalatine branches of the maxillary nerve (frequently referred to as branches of the sphenopalatine ganglion since they come off at this ganglion) enter the nasal mucous membrane through the sphenopalatine foramen and are grouped into lateral and medial posterior superior nasal nerves, the former going to the lateral wall and the latter to the septum, distributing about as shown in Figure 8. The largest of the medial posterior superior nasal nerves is called the *nasopalatine nerve* which ends in the region of the incisive foramen by communicating with the anterior (greater) palatine nerve.

The *posterior inferior nasal nerves*, which distribute to the region of the inferior concha are

branches of the anterior palatine nerve from the sphenopalatine branches of the maxillary nerve. They come off of the anterior palatine nerve as it is coursing inferiorly in the pterygo palatine canal.

Other smaller contributions from the maxillary nerve are the pharyngeal branch of the sphenopalatine to the upper part of the choanal region the nasal branch of the anterosuperior alveolar which enters the mucous membrane of

cous membrane. Also helping to make up the above branches of the trigeminal are efferent fibers carrying impulses to the glands and blood vessels in the lining of the nasal fossa. Some of these efferent fibers are postganglionic thoracolumbar general visceral efferent (sympathetic autonomic) fibers, which are axones of neurones with cell bodies in the superior cervical ganglion which follow the internal carotid artery up into the head and join the

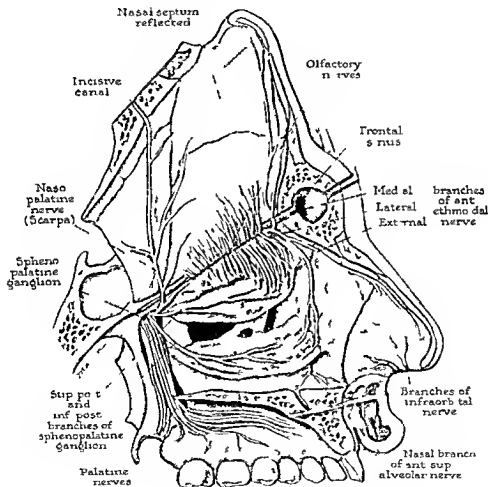


Fig 8 —The nerves supplying the walls of the nasal fossa

the inferior meatus through a small bony foramen in its lateral wall and some small branches of the nasal branch of the infra orbital going to the region of the vestibule.

The branches of the trigeminal nerve described above are for the most part composed of dendrites of afferent neurones with cell bodies in the semilunar (gasserian) ganglion which are carrying impulses of general sensation into the central nervous system from the nasal mu-

trigeminal branches at among other places the sphenopalatine ganglion. Others of the efferent fibers are postganglionic craniosacral general visceral efferent (parasympathetic autonomic) fibers which are axones of neurones with cell bodies in the sphenopalatine ganglion. The preganglionic fibers which synapse with these cell bodies in the sphenopalatine ganglion get there by way of the greater superficial petrosal branch of the facial nerve which together with

the postganglionic thoracolumbar fibers coming to the sphenopalatine ganglion from the carotid plexus (deep petrosal nerve), forms the vidian nerve or nerve of the pterygoid canal. Some general visceral efferent fibers may well distribute to the mucous membrane of the nose by way of periarterial plexuses. Visceral afferent fibers from the nasal mucous membrane are not known well enough to merit description. It is quite generally agreed that thoracolumbar (sympathetic) stimulation produces constriction of the blood vessels but

sphenoid bone, called the *frontal*, *maxillary*, and *sphenoid sinuses*, respectively, while some four to eight in or partly in each lateral mass of the ethmoid bone are called the *ethmoid air cells*.

The communications which these air spaces have with the nasal fossa result from their development as outpouchings of the mucous membrane of the nasal fossa which begin to appear about the third or fourth fetal month, eventually invading the related developing bones, and increasing in size by growth of the

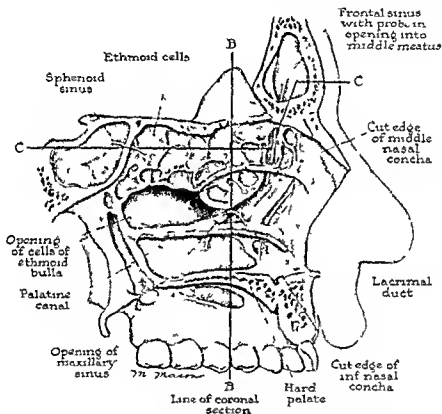


Fig. 9—The paranasal sinuses as seen in a parasagittal section with some bone clipped away to show more of their extent. The plane of this section is indicated by line A' in Figures 10 and 11.

there is not sufficient agreement beyond that point as to the effects of the general visceral efferent (autonomic) innervation of the structures in the nasal mucous membrane of man.

THE PARANASAL SINUSES

The paranasal sinuses or accessory nasal sinuses are air spaces in bones, helping to form the framework of the walls of the nasal fossa, which communicate by relatively small openings with the nasal fossa. There usually are two (right and left) in the frontal bone, one in each maxillary bone, and two (right and left) in the

sac-like outpouchings accompanied by absorption of bone. For the sphenoid sinus, it is probably more correct to think of it as beginning as a partial pinching off of the postero-superior region of the developing nasal fossa.

As one would expect from the development, the sinuses are lined by a mucous membrane which is continuous with the nasal mucous membrane at the ostia of the sinuses. This mucous membrane is similar to the respiratory mucous membrane of the nasal fossa (p. 6), being composed of a ciliated pseudostratified columnar epithelium with some goblet cells and

a fibro elastic tunica propria which is firmly blended to the underlying periosteum so that one refers to a mucoperiosteum here also. The lining of the sinuses is much thinner than that of the nasal fossa and there are fewer glands in the tunica propria. The fact that the mucoperiosteum is thin and that it follows the contours of the bone closely means that unless there is mucous membrane involvement the size and contour of the sinuses are very much the same in the living individual as they would be in the skeleton.

of the sinuses. In a manner similar to that described for the arteries, the nerves supplying the nasal mucous membrane (p 10) to a great extent take care of the lining of the sinuses.

There is no general agreement as to the function or functions of the paranasal sinuses but of the many suggestions which have been made the following seem to merit mention: (1) aiding the nasal cavity in warming and moistening the inspired air (there apparently is air change in the sinuses during respiration), (2) lessening the weight of the head and perhaps giving it better

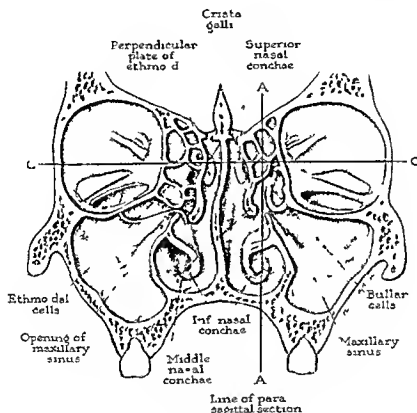


Fig 10—The paranasal sinuses as seen in a coronal section at the position indicated by line B in Figures 9 and 11

The arteries which are supplying the nasal mucous membrane (p 8) also, for the most part, take care of the lining of the sinuses by giving branches to the walls of the sinuses where they lie adjacent to the walls and where they supply the nasal mucous membrane near the sinus orifices. The veins mostly follow the arteries. The lymphatic drainage of the lining of the sinuses has not been definitely determined but it is probable that in general the drainage is by vessels which reach those of the nasal mucous membrane through the orifices

balance, (3) contributing something to the resonance of the voice.

It must be remembered that the following brief account cannot in any way tell the complete story of the anatomy of the paranasal sinuses because of the great variation particularly in the extent to which the sinuses invade the bone and, hence, in their relationship to nearby structures. However, if it is remembered that an individual sinus may spread into any part of the bone in which it is located, even into an adjacent bone that its growth might

have been much less extensive than usual, even to a complete absence, or that one sinus may be encroached upon and even quite completely suppressed by an overgrown adjacent sinus not necessarily of the same group, then a description of the more common conditions can serve as a basis for concept of the paranasal sinuses especially if some indication of the more significant rarer variations is added.

The Frontal Sinuses—Number—There is usually a right and a left frontal sinus, but supernumerary frontal sinuses each with a

observer can rather easily be led to think that the sinus is missing. It must be remembered, as has been indicated in the preceding general discussion of the sinuses, that the frontal sinuses may extend into any part of the frontal bone, the orbital portion being the most common site of extension.

Size—The average measurements of the frontal sinus are about 3 cm in height, 2 to 2.5 cm in width, and 1.5 to 2 cm in depth at the base, and the average capacity is in the neighborhood of 6 or 7 cc.

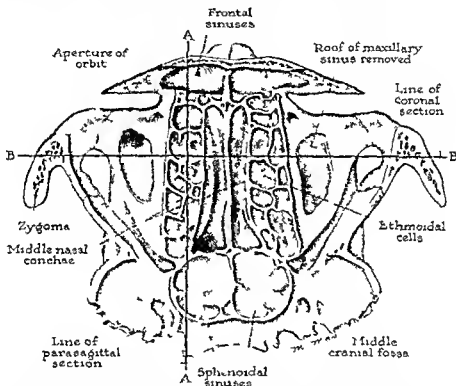


Fig 11—The paranasal sinuses as seen in a roughly horizontal section at the position indicated by line C in Figures 9 and 10

communication with the nasal cavity, are relatively common (about 3 to 5 per cent, Davis). The supernumerary sinuses are readily understood when one remembers that there are several rudimentary outpouchings of the nasal mucous membrane in early development, each of which can invade the frontal bone. Schaeffer has reported a skull with six frontal sinuses. Either or both frontal sinuses may be missing.

Location—The frontal sinuses are usually located one at each side of the midline of the squama of the frontal bone at its junction with the horizontal or orbital portion of that bone. One or both may, however, be in the orbital portion of the bone only, in which case an

Shape—The shape, as usually stated, is roughly pyramidal, but, with extensions into parts of the bone other than the vertical portion, the sinus will, of course, vary greatly from that shape. Also, frequently there are incomplete septa which contribute to an irregularity in the shape.

Dividing Septum—The septum which separates the right from the left frontal sinus is usually 1 mm or less in thickness. Although as a rule it is about in the midline at its anterior inferior end, it seldom runs posteriorly and superiorly from there in the midline and may run very obliquely either to left or right. It is rare for the septum to be incomplete.

Communication with Nasal Fossa—The frontal sinus communicates with the antero superior part of the middle meatus of the nose quite commonly in each of several ways. The opening out of the sinus is usually near the medial end of the floor of the sinus and it may open directly (the more common situation) into the middle meatus or by way of a variable tubular or funnel shaped constriction called the nasofrontal duct which passes between anterior ethmoid air cells and may be encroached on by them. The direct opening or the nasofrontal duct, as the case may be, opens into either the frontal recess (more commonly) or the ethmoidal infundibulum of the middle meatus.

Development—During fetal life there develop several rudimentary outpouchings (the frontal recess, an anterior ethmoidal cell, anterior end of the ethmoidal infundibulum) of the mucous membrane of the anterosuperior part of the developing nasal fossa, any one (more than one in the case of supernumerary sinuses) of which may invade the frontal bone and become the frontal sinus. It is not possible to tell which one will become the frontal sinus until some time after birth. Toward the end of the second year the developing sinus has begun to invade the vertical portion of the frontal bone and by the seventh or eighth year it is beginning to approach the relative amount of invasion of the frontal bone which exists in adult life.

Significant Relations—The chief significant relations of the usual frontal sinus are with the anterior cranial fossa and the orbit. When the frontal sinus empties into the ethmoidal infundibulum and frequently even when it does not, drainage may be taken by the infundibulum to the maxillary sinus.

The Sphenoid Sinuses—*Number*—Most commonly there is a right and left sphenoid sinus. Either one may be quite completely suppressed by the encroachment of the other sphenoid sinus or a posterior ethmoid air cell or there may be failure of growth of one or both. Usually when there are more than two sinuses in the sphenoid bone the extra one is a posterior ethmoid air cell (proved by its communication with the nasal fossa), but there are some substantiated instances of extra true sphenoidal sinuses.

Location—In the "average" condition, one sphenoid sinus is located roughly at each side of the midline in the anterior and anterosuperior part of the body of the sphenoid bone. How-

ever, there may be extension of the sinuses throughout the body of the bone and into any number of its processes.

Size—The average dimensions which are given for the sphenoid sinus are about 2 cm in the longest diameter of each height, width, and length. The average capacity is in the neighborhood of 6 to 7 cc.

Shape—In a very general way the sphenoid sinus could be said to be somewhat cuboidal in shape but it frequently has a very irregular contour because of the crescentic partial septa which are often present and because of extension of the sinus into the processes of the bone and partially around the canals for the optic nerve, vidian nerve, and internal carotid artery which then form ridges on the walls of the sinus.

Dividing Septum—The septum separating the right from the left sphenoid sinus is usually less than 1 mm in thickness and although as a rule, its ventral end is about in the midline it may swing into almost any plane even to a semi horizontal one. Barnhill and Schaeffer both report never having seen a hole in this septum.

Communication with the Nasal Fossa—The ostium of the sphenoid sinus is on the average about 10 to 15 mm above the floor of the sinus on its anterior wall and opens into the sphenoidal recess. The ostium is usually oval, about 2 to 4 mm by 4 to 6 mm for the opening in the mucous membrane, the opening in the bone being larger than this.

Development—The rudiment of the sphenoid sinus is recognizable as early as the fourth fetal month when it is a partially pinched off portion of the posterosuperior part of the nasal fossa in relation to the cartilaginous nasal capsule. A portion of the related part of this capsule ossifies later and becomes attached to the body of the developing sphenoid bone at the second or third year at which time the sinus begins to invade the sphenoid bone. The primitive sinus is now large enough to be pathologically involved. Invasion of the sphenoid bone continues until the sinus reaches its full size.

Significant Relations—Even with a sphenoid sinus of average size there are many structures of importance with which it has relationship, but when the sinus extends to the limits which it sometimes does there are many added relationships of importance. The following are the more important structures which may be in relation with each of the walls of the sphenoid sinus: anterior wall, blood vessels and nerves

crossing the roof of the nasal fossa from the sphenopalatine foramen to supply the nasal septum, lateral wall, cavernous sinus, third, fourth and sixth cranial nerves, ophthalmic and maxillary nerves, internal carotid artery (in carotid canal and in cavernous sinus), ophthalmic artery, and optic nerve (one nerve may be in relation to both sinuses and one sinus may be in relation to both nerves), superior wall, hypophysis and optic chiasma, inferior wall, nerve of the pterygoid canal or vidian nerve, posterior wall, basilar artery and brain stem. It is interesting to note that the sphenoid sinus may be in relation with each of the three cranial fossae and it must be remembered that any of the walls of the sinus may be extremely thin.

The Maxillary Sinus—Number—There is usually one maxillary sinus or antrum of Highmore in each maxilla and there are only a few reported cases of its absence. In the 2 to 3 per cent of maxillae in which two sinuses are found the majority of the extra sinuses prove to be developmentally posterior ethmoid air cells on the basis of their communication with the superior meatus of the nasal fossa.

Location—The maxillary sinus is ordinarily located in the body and zygomatic process of the maxilla but frequently extends to varying degrees into the other processes of the bone.

Size—The average adult maxillary sinus is about 3 cm high, 2.5 cm wide, and 3 cm long and has a capacity of approximately 15 cc.

Shape—The maxillary sinus is roughly pyramidal in shape with the medial wall of the sinus (lateral wall of the nasal fossa) as the base of the pyramid and with the apex in the zygomatic process of the maxilla. At the base there are four sides—the roof, the floor, and the anterior and posterior walls. However, the floor runs only a short distance toward the apex and the anterior and posterior walls meet inferiorly between the lateral limit of the floor and the apex. Close to half of the maxillary sinuses do not have smooth walls but exhibit projections into the sinus ranging from minor ridges to large crescentic partial septa that produce significant pockets which are difficult to drain.

Communication with the Nasal Fossa—The ostium is usually located in the anterosuperior portion of the medial wall of the sinus and opens into the middle third of the ethmoidal infundibulum which communicates with the middle meatus proper by way of the hiatus semilunaris. In one third or more of the in-

stances there are additional ostia which open either into the infundibulum (a rather rare circumstance) or into the middle meatus below the hiatus semilunaris. Both the usual ostia and the additional ostia are located at places where the common wall of the sinus and the nasal fossa is formed entirely by mucous membrane stretched between the bony structures which partly close the large opening in the medial aspect of the body of the maxilla (Fig. 5).

Development—The maxillary sinus begins in the third month of fetal life as an outpouching of the mucous membrane of the ethmoidal infundibulum. By birth it is 7 to 8 mm anteroposteriorly but only about 3 mm in its other diameters. By the end of the first year its lateral growth has almost reached the infra orbital canal which it is beneath at the second year's end. The sinus expands in all directions concurrently with the growth of the maxilla and by the fifteenth to eighteenth years has acquired the adult form.

Significant Relations—The maxillary sinus has five significant relations: (1) The roof of the sinus forms the floor of the orbit and as a rule the canal for the infra orbital nerve forms a ridge on the roof. (2) Typically the roots of the three molar teeth and the posterior portion of the second premolar are in relation with the floor of the sinus where they may produce elevations. With very extensive invasion of the alveolar process all of the teeth would come into relation with the sinus but this is rare. (3) The root of the canine tooth may form a projection on the anterior wall. (4) The superior alveolar nerves are in relation to the anterior and posterior walls of the sinus and contribute to its nerve supply. They are either in bony canals or on the inner side of the bone where removal of the mucoperiosteum will destroy them. (5) From late childhood on, the medial wall is in relation with the middle and inferior meatus but in early childhood the relationship with the inferior meatus is not yet established.

The Ethmoid Air Cells.—Number—There is no constancy in the number of ethmoid air cells. There may be from three to fifteen or more on each side. No description of absence of pneumatization of the lateral mass of the ethmoid bone has been found.

Location—Both the right and left lateral masses of the ethmoid bone are entirely occupied by ethmoid air cells and these extend into the adjacent frontal, lacrimal, maxillary, palatine,

and sphenoid bones at the articulations of these bones with the ethmoid. The articulating bone may just cap over an ethmoid cell or the ethmoid cell may invade the adjacent bone extensively as has been referred to on preceding pages in connection with extra sinuses in the frontal and the sphenoid bones. Air cells usually extend, at least for a short distance, into the supreme and superior conchae and fairly commonly also into the middle concha, agger nasi, and the uncinate process.

Size—Since the ethmoid cells entirely occupy the lateral mass of the ethmoid bone the size of the individual cells depends upon the number of cells present.

Shape—There is so much variation in the shapes of individual cells that it would be of no value to attempt to describe a usual shape.

Dividing Septa—The bony septa between adjacent cells are extremely thin and may be entirely wanting in places.

Communication with the Nasal Fossa—Most observers now prefer to group the ethmoid air cells on the basis of their communication with the nasal fossa. Those which open into the middle meatus are grouped as *anterior ethmoid cells* and are subdivided into frontal cells opening into the frontal recess infundibular into the ethmoidal infundibulum, and bullar (formerly called "middle ethmoid cells") forming the bulla and opening into, for the most part, the supra-bullar recess. The ethmoid air cells which open into the superior and supreme meatuses are grouped as *posterior ethmoid cells*.

Development—By the fourth month of fetal life the outpouchings of nasal mucous membrane destined to become the ethmoid air cells are evident at the sites indicated above where the fully developed cells communicate with the nasal fossa. By the time of birth the ethmoid cells are present as fairly definite entities and they then continue to increase in size until full development is attained.

Significant Relations—The following significant relations are listed: (1) The lamina papyracea, or lateral wall of the ethmoidal labyrinth which separates the ethmoid cells from the contents of the orbit is extremely thin and may even be absent in spots. (2) The anterior and posterior ethmoidal vessels and nerves are in relation to the superior aspect of the ethmoidal labyrinth. (3) There may be absence of bone between the lacrimal sac and anterior ethmoid cells. (4) An anterior ethmoid cell (or cells) may

produce an elevation on the floor of the frontal sinus called the frontal bulla. (5) Posterior ethmoid cells may invade the sphenoid bone in the region of the optic foramen to come into relation with the optic nerve and ophthalmic artery. (6) Ethmoid cells which invade the roof of the orbit come into relationship with the anterior cranial fossa.

JOHN FRANKLIN HUBER

PHYSIOLOGY OF THE NOSE

The physiology of the nose is a subject with which every rhinologist should be thoroughly familiar. The space here available permits no more than the merest mention of the nasal processes which are so important in maintaining the health of the whole respiratory tract.

Beside constituting the natural airway for respiration the nose has three primary functions: (1) it accommodates the olfactory organ, (2) it conditions the inspired air to the requirements of the pulmonary surfaces by warming, moistening, and filtering it, (3) it cleanses itself of the foreign material which it has extracted from the air. This last named function, completely overlooked by most authors, is at least as important as the rest in health, and much more so in the control and eradication of infectious disease. Other attributes ascribed to the nose, such as vocal resonance, gas exchange, and the regulation of air flow, are secondary and usually incidental.

Olfactory Function—The sense of smell in man is only relatively unacute, although classified as a microsmatic animal, man is capable of perceiving certain substances in a dilution of 0.005 micrograms in a liter of air. The end organ of this keenly perceptive olfactory apparatus consists of undifferentiated olfactory cells, uniform in shape, with a spherical nucleus and a peripheral process reaching the surface of the epithelium between so-called sustentacular cells. At the surface the olfactory cell terminates in a bulbous end upon which are numerous olfactory hairs projecting free into the nasal cavity. The central processes of these cells are the axons or olfactory nerve fibers. The olfactory areas are confined to the upper third of the nasal chambers extending from the cribriform

plate onto both the septal and the lateral walls. The olfactory epithelium is pigmented, non ciliated, and merges abruptly with the surrounding respiratory membrane.

Air-Conditioning Function—The inspired air is directed upward toward the roof of the nose by virtue of a constriction one centimeter from the anterior naris. As it reaches the vault, it is deflected sharply backward and downward in a parabolic curve, fanning out as it approaches the posterior naris or choana. The main current passes through the olfactory fissure close to the septum and only an insignificant portion of the air enters the meatuses (Fig 12).

The expired air traverses much the same pathway in the reverse direction except that, owing to the disproportionate sizes of choana and anterior naris, a large eddy occurs which

Inspired air is moistened in passing through the nose to approximately 90 per cent relative humidity and the nasal mucosa has been shown to secrete nearly a liter of water in twenty four hours. Moisture is vital to ciliary activity, and the maintenance of a physiological amount through secretion and the proper humidification of living quarters is a major factor in the prevention of colds.

Filtration of the inspired air is accomplished through the vibrissae in the nostril, through the impingement of air currents against the moist surfaces of the nose, and to some extent through electrical surface charges which cause particulate matter to adhere to the membranes. The dust- and bacteria laden mucus is conveyed to the pharynx through the propulsive agency of the cilia.



Fig 12—Inspiratory air-currents

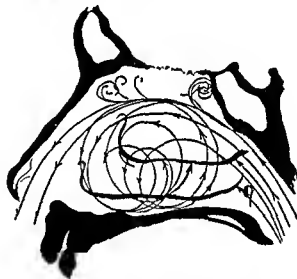


Fig 13—Expiratory air-currents

occupies most of the nasal chamber (Fig 13). In this case, air enters the meatuses both from the choana and from the eddying currents below. Consequently the ostia of the sinuses are protected from the blasts of incoming air and subjected only to expired air which is clean, warm, and moist.

Respiratory pressures fluctuate from approximately +6 mm of water on expiration to -6 mm on inspiration, with the mouth closed. Fluctuations are increased by obstructions about the naris. Pressures within the sinuses naturally fluctuate at the same time and to the same extent unless the ostia are obstructed, in which case fluctuation naturally ceases. Internal pressure may remain above or below the nasal pressures depending upon the conditions under which the closure occurs.

Self Cleansing Function—Considering the multiplicity of infections which can gain access to the body through the respiratory tract it is safe to say that the cilia (together with the overlying mucous blanket) constitute our most important single mechanism of defense. In the normal nose the ciliated epithelium covers the nose in an unbroken sheet from the vestibule to the nasopharynx with the exception only of the olfactory area. A sheet of mucus, thin but cohesive, acts as a conveyor belt, carrying on its surface particulate matter, propelled by the cilia from every part of the nose and sinuses to the pharynx. Ciliary pathways in the sinuses are always toward and through the ostia, in the nose, always toward the pharynx. Those in the bronchial tree are toward the glottis.

Respiratory Cilia—Human respiratory cilia

are approximately 7 microns long and less than 0.3 micron in diameter. They are packed closely on the free surface of the epithelial cell and beat from eight to twelve times a second. Each cycle consists of a rapid "effective" stroke and a slower "recovery" stroke.

Cilia are primitive structures and hence extremely viable. They beat many hours after death, in fact until the disintegration of the epithelium itself sets in, they may continue to beat vigorously in the presence of infection, inundated with blood and pus, and they are commonly regenerated together with the rest of the epithelium following the stripping of the mucosa from the bone. They are slowed down by cooling and warming, the optimal temperature being 32°C and although they cease beating when the temperature is reduced to 10°C , they resume their motion with momentarily increased activity on rewarming. Drying is fatal to them in a very few minutes.

Preserving the integrity and maintaining the activity of these microscopic structures and at the same time keeping the overlying mucous blanket at its physiological best constitute a fundamental requirement of nasal surgery and therapy.

Ventilation and drainage have always been recognized as the cardinal requisites of nasal health, to these the modern concepts of physiology compel us to add moisture.

ARTHUR W. PROETZ

DERMATOSES OF THE EXTERNAL NOSE

Involvement of the skin of the nose by disease is in the majority of cases only a very small part of a much more widespread involvement of the cutaneous envelope. For example, seborrheic eczema, acne vulgaris, and acne rosacea may appear prominently on the nose but usually limited or extensive areas elsewhere show the same disease. With a few diseases the lesion or lesions may be limited to the nose as in furunculosis, lupus erythematosus, lupus vulgaris, syphilis, senile keratosis, and carcinoma, but with these conditions lesions also may be found elsewhere.

Impetigo Contagiosa—Impetigo contagiosa is an acute, contagious, inflammatory disease of the skin, characterized primarily by superficial, discrete, slightly elevated vesicles which quickly become pustular, rupture, and dry on the skin as thin crusts. Usually the disease is of staphylococcal origin, although occasional cases are reported to be due to streptococci.

Symptoms—The lesions may occur upon the external surface of the nose but are more common at the nares and may extend into the vestibule. They begin as barely elevated vesicles or bullae which become vesicopustular or pustular within twenty-four hours. Rupture quickly occurs and the exudate dries upon the skin as a thin, yellowish crust which is usually easily detached or may drop off exposing a reddish, moist spot.



Fig. 14—Impetigo of the nose

Diagnosis—There is no other dermatosis of the nose which closely resembles impetigo contagiosa.

Treatment—The crusts should be removed from lesions over the nose by means of wet compresses of a saturated solution of boric acid or by moistening thoroughly with an antiseptic oil. A bactericidal agent is then applied directly to the exposed moist surface. An ointment, containing either 75 or 150 grains (5 or 10 gm) of ammoniated mercury or 25 to 50 grains (1.6 to 3.2 gm) of sulfathiazole to the ounce of aquaphor, applied at three-hour intervals, is usually effective in causing the disappearance of the lesions within three to seven days.

Harris¹ has recommended the application of a solution of microcrystalline sulfathiazole directly to the moist lesions, which are then covered with a gauze pad. Cure within twenty-four

hours in some cases is reported. If cure with a sulfonamide is not complete within a week, some other drug should be substituted because of the danger of sensitization. At the nares, painting with a solution of 1 per cent each of acriflavine and gentian violet is often quickly effective, although unsightly. Roentgen ray therapy may aid in the treatment of resistant cases, but doses should be fractional (50 to 90 units at intervals of three to four days).

Erysipelas—Erysipelas, also known as St. Anthony's fire, is an acute inflammation of the skin and subcutaneous tissue, characterized by a deep red color, swelling, heat, pain, and vesiculation, and accompanied by fever and constitutional disturbance. The exciting cause is the streptococcus of Fehleisen. The organism may find entrance through any break in the skin.

Symptoms—The disease may start on the nose and extend to involve the entire face. It first appears as a localized, red elevation which extends peripherally with an elevated border. The area is hot and tender to the touch. Constitutional symptoms are chill, malaise, headache, and elevation of temperature (102° to 105° F). There is a mild recurrent form of erysipelas which attacks the alae of the nose and the cheeks. Constitutional disturbance with this type is mild or absent. The eruption does not spread beyond the face and usually disappears in three or four days.

Diagnosis—Erysipelas is to be differentiated from an acute dermatitis due to an external irritant. Fever and constitutional symptoms are lacking in the latter.

Treatment—Siegel, Rosove, and Bower² consider sulfonamide drugs the treatment of choice, although they report a mortality of 1.3 per cent in 303 patients treated with sulfamamide. They recommend 1 grain (0.065 gm) per pound weight per day for children up to five years of age, giving half the dosage at once and the remainder in four equal doses. In older patients the plan of treatment is similar except that dosage is 15 grains (0.975 gm) per 20-pound body weight daily. Local treatment consists of cold wet compresses of a saturated magnesium sulfate solution. Roentgen ray therapy given in 100 r unit doses each day for three days is often rapidly effective.

Prognosis—With sulfonamide therapy a mortality of from 1.3 to 3 per cent is reported. In rare cases, abscesses or gangrene may result.

Furuncle—A furuncle is an acute circumscribed inflammation of a hair follicle or sebaceous gland ending in suppuration and the expulsion of a central necrotic mass. Staphylococcus pyogenes aureus is responsible.

Pathology—There is a dense leukocytic infiltration around a sebaceous gland or hair follicle with thrombotic obstruction of the capillary blood vessels and central necrosis.

Symptoms—A furuncle begins as a deep-seated induration. Occurring on the nose, the most common site is the tip. A reddish, rounded prominence is visible and this area gradually softens as a central core forms, evidenced by a yellowish color. Pain may be intense.

Complications—A sudden chill and elevation of temperature in association with headache and edema of the eyelids may signify sinus thrombosis.

Treatment—Great care and caution are necessary in treating a furuncle of the nose because of the danger of spreading the infection to the orbit and cavernous sinus. The affected individual must be warned not to pick or squeeze the lesion. A beginning lesion may be aborted by a roentgen ray application of 200 r units unfiltered. Sulfadiazine in a dosage of 15 grains (0.975 gm) every six hours should be given at once. Frequent or continuous hot compresses of a saturated solution of boric acid should be applied to more advanced lesions until softening and pointing are complete. At this time light pressure with a sharpened wooden applicator dipped in a solution of phenol will result in rupture and promote drainage. The use of staphylococcal toxoid, or stock or autogenous vaccine aids in raising the patient's resistance in recurrent furunculosis.

Lupus Vulgaris—Lupus vulgaris is a tuberculous new growth, characterized by reddish or reddish-brown patches composed of papules, nodules, and flat infiltrations, usually terminating in ulceration and scarring. Invasion of the skin by the tubercle bacillus is the cause.

Pathology—Histologic study of a section of lupus tissue shows sharply circumscribed nests of cell infiltration in the deeper layers of the corium. Epithelioid cells are present, and there are many giant cells. Tissue from ulcerated areas shows coagulation necrosis and fatty degeneration of the center of the nodules. Bacilli are present but difficult to find.

Symptoms—The disease begins as one or several tiny, reddish or reddish brown papules.

which under glass pressure show an "apple jelly" color. The papules gradually enlarge into nodules (tubercles) which group to form an irregular patch. The nodules may disappear leaving a scarred, scaly skin or may ulcerate, crust, and eventually heal with scarring.

Diagnosis—In the differential diagnosis, tertiary syphilis is chiefly to be considered. Nodules of syphilis are hard, the ulcers deep and punched out, and the scars whitish, soft, and smooth. Nodules of lupus vulgaris are soft, the ulcers superficial with soft undermined edges, and the scars are yellowish, shrunken, and hard. Rhinoscleroma may be ruled out by its



Fig 15—Lupus vulgaris

relative rarity in the United States. Epithelioma only slightly resembles lupus vulgaris.

Complications—Carcinoma may develop in a patch of lupus vulgaris in from 1 to 2 per cent of the cases.

Sequelae—Scarring is inevitable. Occasionally the nose is destroyed.

Treatment—General treatment consists of a nutritious, high vitamin diet. The Gerson-Hermansdorfer diet, which is essentially low protein, low carbohydrate, high vitamin, and salt free, is believed to stop the progress of the disease. Local treatment must be heroic to be effective. Under general anesthesia thorough curettage followed by cauterization is considered a valuable procedure. Superficial patches may be scarified by numerous parallel incisions, crossed at right angles by others, and followed by the application of a 1:4000 solution of bichloride of mercury.

Syphilis—Syphilis is a chronic infectious and contagious disease, either acquired or congenital, intermittent in character, and capable of infecting or affecting every structure of the body. The *Spirochaeta pallida*—a spiral organism varying from 4 to 20 microns in length and having from 6 to 20 spirals—is the causative organism.

Pathology—The syphilitic process is characterized by a distinctly circumscribed and homogeneous cell infiltration, tending to spread on the periphery, at the same time undergoing central involution. The cell infiltration exhibits a characteristic tendency to surround and involve blood vessels and lymphatics. The infiltrate, which lies in the corium and subcutaneous tissue, disappears either by absorption or ulceration.

Symptoms—Chancre of the nose is almost unknown although theoretically possible. Approximately six weeks following the appearance of the chancre, or primary lesion, there occurs in a majority of patients an explosion of lesions over every part of the body. These lesions may be macular, papular, or pustular, but not vesicular. In the course of involving the entire cutaneous surface the nose may be involved. Recurrent secondary lesions usually are annular and commonly occur about the alae of the nose. Late or tertiary syphilis develops slowly and insidiously. The nose may be involved with nodular or nodulo-ulcerative gumma. Nodules, when present, are firm and painless. Ulcers are deep, with sharp-cut edges and when healed leave soft, smooth, whitish or pigmented scars. The nares may be involved in infantile congenital syphilis with mucous patches which extend into the nasal cavity and give rise to the snuffles. In "tardive" or late congenital syphilis destruction of the bones may give rise to a depressed or so-called "saddle" bridge.

Diagnosis—In secondary syphilis diagnosis is confirmed by the typical eruption elsewhere over the skin and evidence of a healed primary lesion. Tertiary syphilis of the nose is to be differentiated chiefly from lupus vulgaris.

Sequelae—Tertiary syphilis is followed by a disfiguring scar.

Treatment—In the treatment of syphilis, in the secondary stage, the rule of the Cooperative Clinical Group of the United States Public Health Service should be followed—namely, to give at least 30 injections of an arsenical and 60 injections of a heavy metal, preferably bismuth.

with no rest periods. The arsenical may be old arsphenamine (606), neoarsphenamine, or mapharsen. The doses are as follows:

	INITIAL DOSE	SUBSEQUENT DOSE
Arsphenamine	Female 0.3 gm	0.4 gm
	Male 0.3 gm	0.45 gm
Neoarsphenamine	Female 0.3 gm	0.45 gm
	Male 0.45 gm	0.6 gm
Mapharsen	Female 0.03 gm	0.045 gm
	Male 0.04 gm	0.06 gm

Bismuth should preferably be of the oil-soluble type because in this form it is rapidly absorbed and slowly excreted. The doses are 2 grains (0.13 gm) for the female and 3 grains (0.195 gm) for the male. During the first fifteen weeks an intravenous injection of an arsenical and an intramuscular injection of bismuth may be given conjointly each week, followed by fifteen weeks of bismuth and finally fifteen



Fig. 16—Gumma of the nose (tertiary syphilis)

weeks of bismuth alone. This adds up to the required totals for both the arsenical and the heavy metal.

Although the ultimate worth of penicillin has not been determined, its rapid effects suggest that it may eventually supplant or be incorporated into one of the present therapeutic systems. Its present usage consists in the intramuscular injection of 20,000 Oxford units every three hours, day and night, until a total of 1,200,000 units has been given. This course may be repeated.

In tertiary syphilis, treatment may be initiated with iodides by mouth and intramuscular bismuth, resorting to the arsenicals later if the patient is below the age of fifty-five. Dosages for late syphilis in older persons should correspond to those given above for females.

The manifestations of infantile congenital syphilis respond promptly to almost any type of antisyphilitic therapy. A nursing child will often receive enough arsenic through the milk of a mother, who is receiving arsenical therapy, to act therapeutically. For bottle babies 1 gram (0.065 gm) of mercury with chalk added to each bottle is efficacious. According to Hinrichsen³ acetarsone (stovarsol), given by the low intermittent dosage scheme, is the ideal drug for infantile congenital syphilis. The succeeding schedule of treatment as given by Kampmeier⁴ may be followed.

SCHEDULE FOR THE TREATMENT OF FLORID CONGENITAL SYPHILIS PARTICULARLY IN EARLY INFANCY

Time	Drug
1st week	0.002 gm acetarsone* per lb of body wt daily
2nd week	0.005 gm acetarsone* per lb of body wt daily
3rd week	0.007 gm acetarsone* per lb of body wt daily
4th through 10th week	0.01 gm acetarsone* per lb of body wt daily
11th through 14th week	1 gm mild mercurial ointment† daily‡
15th week	0.005 gm acetarsone* per lb of body wt daily
16th through 24th week	0.01 gm acetarsone* per lb of body wt daily
25th through 28th week	1 gm mild mercurial ointment† daily‡
29th week	0.005 gm acetarsone* per lb of body wt daily
30th through 38th week	0.01 gm acetarsone* per lb of body wt daily
39th through 42nd week	1 gm mild mercurial ointment† daily‡
43rd week	0.005 gm acetarsone* per lb of body wt daily
44th through 52nd week	0.01 gm acetarsone* per lb of body wt daily
53rd through 56th week	1 gm mild mercurial ointment† daily‡

* Acetarsone is given by mouth. The drug should be dissolved in a portion of the child's formula or other liquid food. The day's dosage may be given at one time.

† The mild mercurial ointment should be rubbed into a new skin site immediately following the daily bath.

‡ Substitute weekly injections of 0.03 gm bismuth subsalicylate in oil intramuscularly whenever it is possible to carry out this treatment.

In older children with tardive syphilis, alternate courses of neoarsphenamine and bismuth, until the child has had a total of 30 of the former and 60 of the latter are recommended. It

is suggested that the doses of neoarsphenamine according to age are as follows Age 7—0.2 gm, age 10—0.25 gm, age 12—0.3 gm, age 15—0.4 gm Bismuth may be given in doses of 1 to 2 grains according to age and weight

Lupus Erythematosus—Lupus erythematosus is an acute or chronic disease characterized by circumscribed reddish patches at times covered by adherent scale, and usually tending to produce scarring of the affected skin The cause of this disease is unknown, although acute patches may result from overexposure to the sun

Symptoms—Early lesions consist of slightly elevated, rounded or oval, sharply margined, pinkish or reddish spots When present on the nose, extension peripherally onto the cheeks often occurs giving rise to a butterfly shaped



Fig 17—Lupus erythematosus with scarring of the nose

patch Adherent whitish scales are present on the surface, the removal of which discloses patulous and distended openings of sebaceous glands Ulceration never occurs

Diagnosis—The absence of nodules and ulceration makes the differential diagnosis between lupus vulgaris and syphilis very easy Secondary dermatitis does not cause scarring

Sequelae—Scarring commonly occurs in a patch of lupus erythematosus Carcinoma may develop in old patches

Treatment—Prolonged exposure to sunlight must be avoided Gold salts and bismuth are the drugs of choice but gold salts are not to be used in acute cases or in patients with a leukopenia Treatment is best started with intramuscular injections of bismuth subsalic

ylate or subsalicylate in doses of 2 to 3 grains (0.13 to 0.195 gm) weekly If gold salts are used a preparation of choice is gold and sodium thiosulfate, given intravenously beginning with a dose of 5 or 10 mg dissolved in 5 cc of sterile distilled water Subsequent doses may be gradually increased to a maximum of 50 mg Gold therapy is to be stopped immediately if the patient complains of generalized pruritus or develops a scarlatiniform rash

Prognosis—With modern therapy many patients recover completely Recurrences are not uncommon but respond to further therapy Death may occur if the disease becomes disseminated

Acne Vulgaris—Acne vulgaris may involve the nose as part of a more widespread involvement of the face Comedones, papules, and pustules may be present

Rosacea—Rosacea, also known as acne rosacea and at times referred to as 'brandy nose' is a chronic hyperemic disorder of the face particularly of the nose and cheeks, characterized by redness dilation of the blood vessels the formation of papules and pustules, and in some cases by connective tissue hypertrophy Common causes of rosacea are chronic gastrointestinal disturbances, dietary indiscretions including the excessive use of alcoholic beverages, menstrual and uterine disorders, and continued exposure to cold winds Klaber and Wittkower⁵ believe that rosacea often results from emotional disturbances, which act directly by producing 'permanent blush' or perhaps more often by lowering gastric tone which may lead to a permanent flush Ayres and Anderson⁶ report the finding of the Demodex folliculorum in large quantities

Pathology—There occurs a dilatation of the blood vessels followed by permanent enlargement and connective tissue overgrowth In hypertrophic cases the corium is greatly thickened and the sebaceous glands somewhat enlarged

Diagnosis—Lupus vulgaris and tertiary syphilis may simulate acne rosacea, but both tend to ulcerate, whereas acne rosacea does not The presence of hyperemia, with enlargement of the vessels, and acne papules and pustules, occurring in the nose and cheeks and running a chronic course, renders the diagnosis easy

Symptoms—The disease may go through three stages In the first stage there is a diffuse redness of the affected part, commonly the nose, which is aggravated by eating, drinking

alcoholic beverages or hot tea or coffee, exposure to cold or heat, or nervous excitation. After several months or years, papular and pustular lesions appear marking the beginning of the second stage. The redness is now more persistent and fine capillaries and vesicles may be visible. If the disease progresses to the third stage the capillary enlargement becomes marked and there is a hypertrophy of the sebaceous glands and connective tissue which may lead to a bulbous enlargement of the nose (rhinophyma).

Sequelae—Permanent enlargement of the surface blood vessels and hypertrophy of the affected tissues may result.

Treatment—The cause or causes of the disease must be sought and eliminated. It is always well to place the patient on a diet restricting excess carbohydrates, condiments, sweets, alcohol, tea, and coffee. The bowels should be regulated. If the patient has symptoms that may be attributed to the menopause, appropriate therapy, usually consisting in the injection or injections of estrogenic substances, should be instituted. The causes of emotional disturbances should be sought and eliminated whenever possible. If the problem cannot be solved immediately, phenobarbital ($\frac{1}{4}$ grain—0.16 gm—per dose) may be administered three times daily for a brief period of time but bromides should be avoided.

Local treatment is at times invaluable. One of the best topical applications is Kummerfeld's solution, the formula for which follows:

R Precipitated sulfur	1 to 4 drachms (4 to 16 gm)
Powdered camphor	10 grains (0.65 gm)
Powdered tragacanth	20 grains (1.3 gm)
Lime water	2 fluid ounces (60 cc)
Rose water	2 fluid ounces (60 cc)

Roentgen ray therapy in small fractional doses (30 to 60 r units weekly) is often of value but may cause a temporary exacerbation in the skin outbreak. When the capillaries are enlarged they may be destroyed by scarification or by electrolysis, application being made for three or four seconds to each vessel. In hypertrophic cases ablation of the diseased tissue may be performed by electrodesiccation or with the cutting current.

Granuloma Rubra Nasi—Granuloma rubra nasi, which was called by Lushen "a peculiar form of acne with changes in the sweat glands," is a chronic inflammatory disease of the skin

covering the cartilaginous portion of the nose. The disease usually occurs in children between the ages of six months and sixteen years. Heredity may play a role but the exact cause is unknown.

Pathology—The corium shows inflammatory changes with dilatation of the blood vessels, and an infiltration of leukocytes, plasma and mast cells, and connective tissue cells.

Symptoms—The skin of the nose shows a fairly well defined red area, on which are scattered pinpoint- to pinhead-sized dark red macules and papules. A characteristic feature is the presence among the macules and papules of beads of perspiration. Vesicles may be present. The nose is cold to the touch but there are no subjective symptoms.

Diagnosis—The disease slightly resembles lupus vulgaris, but the absence of ulceration and the disappearance of the lesions under glass pressure make the differential diagnosis easy.

Treatment—There is no effective therapy, but the disease disappears during the period of puberty. Dusting the area with tannin powder has been recommended as a local measure.

Senile Keratoses—A senile keratosis is a slightly elevated, brownish or black lesion covered with thin scales or crusts, beneath which is a surface that bleeds easily and may be superficially ulcerated.

Treatment—A lesion of this type must be thoroughly destroyed to prevent malignant degeneration. Cure may be accomplished by roentgen ray or radium therapy, an erythema dose of either often being sufficient. A more rapid and definite cure is accomplished by curetting away the lesion under local anesthesia and desiccating the base. Carbon dioxide snow applied from fifteen to forty seconds with moderate pressure is also curative.

Prognosis—A senile keratosis may remain benign for periods of time varying from months to years, but the danger of the development of a basal cell or squamous cell carcinoma always exists.

Epithelioma—An epithelioma may be basal cell, basal-squamous cell, squamous cell, or melanotic. The cause of cutaneous cancer is unknown although trauma and repeated irritation are predisposing factors. Epithelioma of the nose often results from wearing pince nez.

Pathology—The essential process in epithelioma is the proliferation of epithelial cells and

their extension in structures not normally the seat of these cells. In the infiltrated areas, mitotic figures are seen. The tumor cells in the pigmented type of epithelioma are laden with melanin.

Symptoms—Early superficial lesions may appear as small, pearly or waxy nodules or as warty excrescences. The lesions gradually extend peripherally and in depth. Ulceration of the center eventually occurs and this area becomes crusted. Removal of the crust or injury to the lesion causes bleeding. The border is elevated, rolled out, and has a shiny or waxy appearance. If allowed to develop the ulceration in epithelioma of the nose may eventually penetrate through the nose.



Fig 18—Early epithelioma of the nose

In squamous cell lesions of the nose the growth is more rapid than in the pure basal cell type. Squamous cell lesions are also more prone to metastasize.

Treatment—A basal cell epithelioma may be destroyed by roentgen ray, radium, or electrodesiccation, or may be removed surgically. Roentgen rays must be administered in doses varying from 700 to 1400 r units and radium in a dose sufficient to produce a local reaction. Electrodesiccation under local anesthesia is the surest way of obtaining a complete cure. The method consists of desiccating the lesion superficially, followed by curettage of all the tissue that will come away, and then applying electrodesiccation to the base. A simple calamine lotion or a surgical dusting powder may then be applied to form a protective crust. Healing re-

quires from three to six weeks depending upon the depth of the growth. Squamous cell growths are often radioresistant and should be thoroughly destroyed.

Prognosis—If the lesion has penetrated to the mucous membrane plastic surgery is usually required. In the majority of cases, epithelioma of the nose may be cured if seen early and thoroughly destroyed. If penetration through the nose occurs the prognosis is poor.

CARROLL S. WRIGHT

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RHINITIS

ACUTE RHINITIS

Synonyms Common Cold, Acute Infection of Upper Respiratory Tract, Acute Coryza

Definition—An acute inflammation of the mucous membranes of the nose characterized by swelling and edema and accompanied by nasal irritation, sneezing, rhinorrhea, and malaise.

Etiology—*Virus Infection*—That an ultra-microscopic filtrable virus is capable of producing acute rhinitis was shown by Kruse in 1914. Supporting evidence has accumulated since that time, and the virus has been cultivated in special media in vitro. The transmission of colds to men and anthropoid apes by this virus was successful in approximately 40 per cent of attempts. Of significance among the bacteriologic findings in individuals so infected is the increase in the number of the potentially pathogenic bacteria which are normally present in

the nose and throat. Colds produced by means of the virus under experimental conditions of strict isolation were characterized by profuse rhinorrhea, sneezing and malaise, and a gradual return to normal in three to seven days. Under conditions of ordinary life where there is exposure to other pathogenic bacteria, secondary infection leads to a purulent rhinitis and the common sequelae of sinusitis and bronchitis.

Bacterial Infection—The entrance of pathogenic bacteria through the epithelium of the nose to the subepithelial tissues gives rise to acute rhinitis. The most common offenders are streptococci, pneumococci, staphylococci, *Bacillus influenzae* and *Micrococcus catarrhalis*.

The evidence at hand serves to indicate that the infection with the virus is a short, self-limiting disease, and that the invasion of the tissues with pathogenic bacteria constitutes the main etiologic factor in acute rhinitis.

Pathology.—The changes found in the nasal tissues are those of simple inflammation. There is a dilatation of the glands of the mucosa and of the goblet cells in the epithelium. There is dilatation of the blood vessels. Some desquamation of the epithelium with loss of ciliated cells is present in the infections caused by the virus. Infiltration of the tissues with polymorphonuclear leukocytes is present.

Predisposing Factors—Factors which disrupt the integrity of the normal nasal defense predispose to the entrance of bacteria to the subepithelial tissues. Factors which lower the normal resistance of the individual predispose to the growth of bacterial invaders in the tissues.

The normal defenses of the nose are (1) the blanket of mucus overlying the epithelium, (2) the cilia which move this mucus. Sticky mucus entangles foreign particles, such as bacteria, and conveys them to the nasopharynx and pharynx where they are swallowed. Essential for the maintenance of the defense are the continuity of the mucus blanket and the activity of the cilia to move it. Thus, factors which impede ciliary action or which disrupt the mucus blanket predispose to the entrance of organisms to the subepithelial tissues. Such factors may be considered as (a) physical or (b) metabolic.

PHYSICAL FACTORS—Cilia will function actively, even under abnormal conditions, provided they are kept moist. If they become dry, activity soon diminishes and in a short time ceases. Cilia may be resuscitated by further

moistening. Cilia function actively in temperatures from 18° to 40° C., at either extreme they beat more slowly and finally stop.

These facts are pertinent in consideration of factors which destroy defense mechanisms. The dry atmosphere commonly encountered in over-heated rooms in wintertime tends to slow ciliary function and to make the mucus in the nose more viscid and crusty. An area of crust on the mucous membrane is a potential site for entrance of bacteria to the underlying tissues. Drying of areas in the posterior nares or in the nasopharynx may be caused by the diversion of air currents by anatomic abnormalities. Deviations and spurs of the septum, for example, may deflect air currents so that they impinge on one small area in the posterior part of the nose and cause drying.

METABOLIC FACTORS—The natural immune processes of the body termed "resistance" consist of the vasomotor response of the tissues, the activity of the phagocytic cells, and the presence of antibodies in the blood and tissues. All such processes are dependent upon the maintenance of a normal body metabolism. Factors such as fatigue, exposure, dietary excesses or deficiencies, and endocrine dysfunctions, alter the normal metabolic processes and lower resistance. Organisms gaining entrance to the subepithelial tissues under such conditions may grow and spread unchecked.

The vasomotor changes incident to excesses in alcohol intake and dietary indiscretions cause swelling of the turbinates and alterations in air flow through the nose with consequent drying in the nasopharynx. Allergic changes also may cause such alterations and infections.

Symptoms—General Prodromal—In those cases in which the infection is due primarily to a breakdown in the normal defense mechanisms the stage of onset is preceded by a period of irritation in the nasopharynx lasting about twenty-four hours. The patient complains of a dry, burning sensation above the soft palate which may be accompanied by some malaise and headache. This is due to the drying of a small area of mucosa in the nasopharynx and the formation of crusts with consequent inflammation of the surrounding mucous membrane. Examination at this time frequently reveals a small hyperemic area in the nasopharynx while the mucosa of the nose and pharynx is essentially normal. In those cases in which the virus

is the etiologic factor the patient complains of a sudden onset of malaise, chilliness, and usually headache

After a period of perhaps twelve to twenty hours the nose becomes obstructed and this is followed by nasal irritation, sneezing, lacrimation, and a profuse watery nasal discharge

Acute Stage—The temperature rarely rises above 99° F (37.8° C) There is tendency toward leukopenia, and the white blood cell count may be about 8000 per cc At this time the nasal mucous membrane is diffusely red, swollen, and wet, the nose is filled with clear watery secretion, the pharyngeal and nasopharyngeal mucous membranes may also be diffusely hyperemic

Stage of Resolution—The duration of the acute stage varies from two days to a week and is followed by the stage of resolution The character of the nasal discharge changes and instead of profuse watery secretion the discharge becomes purulent, nasal obstruction is more marked, and postnasal discharge may cause laryngitis and cough The color of the mucous membrane becomes less angry red, and gradually, after perhaps a week to three weeks, the nose returns to normal

Diagnosis—The common cold must be differentiated from the acute rhinitis associated with the exanthemas The appearance of the rash in scarlet fever and the presence of Koplik's spots and conjunctivitis in measles, for example, make the diagnosis possible In the early stages, influenza may cause difficulty, but the more generalized symptoms in the latter condition, with muscular aches and with the scarcity of nasal symptoms, make differentiation possible Frequently confused with acute infectious rhinitis is acute nasal allergy The history of previous allergic upsets—nasal or otherwise—together with the presence of eosinophils in smears of the nasal secretions and general pallor of the mucous membranes differentiates allergic reactions from infectious rhinitis The mucosa in allergic rhinitis may at times be of the same fire red color as that of infectious rhinitis, but a high percentage of eosinophils in the nasal secretion is diagnostic of allergy

Complications—Acute infection of the nasal mucous membrane may spread to adjoining organs It is usual with any infection of the nose to have concurrent infection in the nasal accessory sinuses When the structural rela-

tion of the accessory sinuses and the continuity of their mucous membranes with that of the nose are considered, it is not surprising that there is some degree of pansinusitis with each attack of rhinitis The sinus infection usually subsides with the subsidence of the rhinitis in adequately treated cases

Spread of infection by way of the eustachian tubes may lead to infection of the middle ear and mastoid air cells This is more apt to occur in instances in which excessive and improperly performed nose blowing is practiced The aspiration of secretions from the nasopharynx may lead to infection of the larynx and to tracheitis and bronchitis, and occasionally to pneumonia

Treatment.—Prophylaxis—The common cold, acute rhinitis, is probably the greatest single cause of loss of time from productive occupation This has been emphasized recently in war industries and in the armed forces For this reason much emphasis is placed on the prevention of this disease The problem of prevention should be considered from two viewpoints (1) measures whereby the chance of infection is lessened, (2) measures to increase the resistance of tissue to infective micro-organisms

REDUCING CHANCE OF INFECTION—The importance of adequately humidified air in living rooms and in bedrooms cannot be overemphasized Patients should be urged to keep the temperature of rooms between 68° and 70° F, and to keep the humidity at about 45 per cent of saturation The maintenance of adequate humidity is not easily accomplished Humidifiers are helpful In apartments, pans of water on the radiators help, even steam from a kettle may prove useful at night Although it is commonly considered necessary to have the windows open at night this practice should be used with caution The cold, dry air in winter may be excessively drying in the nose, or through vasodilatation and engorgement of the turbinates, may lead to mouth breathing and pharyngitis Clothing is most important in relation to respiratory infections in children If the undergarments are woolen and heavy the active child will perspire freely in the warm indoors Unless he is adequately protected when he goes outdoors he is apt to chill and infection will follow Light weight underclothes, preferably of cotton, are advisable for children, with warm coats, shoes, et cetera, for the outdoors

The general health depends on adequate, well-balanced diet. Additional vitamins for children and adults prove helpful but do not take the place of a properly distributed food intake. The prevalent habit among so many working women of consuming a cup of black coffee for breakfast and a sandwich and ice cream for the hurried lunch leads to fatigue and predisposition to infections. Insistence on proper eating habits will do much to prevent infections of the upper respiratory tract.

Fatigue lowers the general resistance to infection. Adequate hours of sleep are important in the prevention of infections, and periods of rest during a busy day do much toward preventing fatigue and improving efficiency.

The avoidance of crowded public places, where, at best, ventilation is inadequate, should be urged during epidemics of respiratory infections.

INCREASING TISSUE RESISTANCE —Vaccines — For years, vaccines of the common respiratory pathogens have been advocated as a means of prophylaxis against acute rhinitis. D and R. Thompson,¹ surveying the literature prior to 1932 say, "In our opinion there is abundance of irrefutable evidence in favor of the value of vaccines, both toxic and detoxicated, in the treatment of colds and catarrh of the upper respiratory tract. Preventive inoculations against colds give no guarantee of immunity to catarrh, but it is our experience that colds contracted by an inoculated person are less severe as a rule than those contracted by the uninoculated, furthermore, the inoculated are less liable to complications." Von Sholly and Park,² however, who gave injections of vaccine to 1536 persons and had as controls 3025 uninoculated persons state "On the whole our evidence does not make a strong case in favor of the vaccine given by us as a prophylactic agent against acute respiratory diseases—pneumonia *albane* excepted." Jordan and Sharp³ had some 6000 persons under observation in 1921. One half were vaccinated, the rest were untreated. They conclude "Rhinitis and bronchitis developed with about equal frequency in the vaccinated and unvaccinated groups." Blitch and Doyle⁴ used a stock vaccine for the prevention of colds in soldiers at Fort Monmouth, N. J., in 1936-37. They vaccinated 375 men and had 475 as controls. The total admission to sick report (rate per 1000 men) was 209 for the vaccinated and 224 for the unvaccinated. The

average days lost from duty per man was 5.9 for the vaccinated and 6.1 for the unvaccinated. They conclude that vaccination does not prevent colds or their complications nor does it cut down the number of days lost because of colds.

The results of vaccination against colds by subcutaneous and oral methods are disappointing. The question arises: Wherein lies the cause of failure? Is it that the upper respiratory pathogens are poor antigens, or is it perhaps that in spite of an increase in circulating antibodies against the pathogens the portal of entry of these organisms is still left unguarded? It is generally agreed that the ideal conditions for tissue immunity are those in which there are not only sufficient specific antibodies but also an accumulation of phagocytes in the tissue to be guarded. Walsh and Cannon⁵ showed that in the nose such a condition can be effected by the simple application of a vaccine to the nasal mucosa. They found that following the local application of a vaccine of paratyphoid bacillus in the nose of rabbits the agglutinin titer of the nasal tissues was frequently as high as that of the blood serum and constantly higher than that of other tissues such as spleen, liver, and gut, also that the nasal tissues of animals so vaccinated showed a subepithelial accumulation of macrophages and a hyperplasia of lymphoid tissue. These changes were not present when the vaccine was administered by subcutaneous injection or by mouth. They also showed that in animals immunized against two different antigens simultaneously, the ratio of tissue antibodies to serum antibodies (T/S ratio) was constantly higher for the locally applied antigen than for that administered generally. On the average the T/S ratio for parenterally administered antigens is from 1/10 to 1/15, whereas that for locally applied antigens is 1/5. This accumulation of specific antibodies in an area in which there is an accumulation of phagocytic cells enhances the resistance of tissue and should constitute an adequate barrier against the invasion of pathogenic microorganisms. That such an increased resistance to infection is obtained is shown by Walsh and Cannon's experiments and those of Bull and McKee⁶ and Linton⁷ in which, following nasal vaccination, there occurred an increased resistance to intranasal infection by a virulent organism, in spite of the fact that no specific antibodies could be demonstrated in the blood stream.

Treatment.—The objective of treatment is the restoration of the nasal airway. Infection in the sinuses, if it is present, must be eliminated. Allergy, when present, should be treated. Attention should be given to the diet. Adequate vitamin and protein intake and a reduction in carbohydrates are advisable. In many cases the administration of thyroid extract produces marked improvement. There is hope that the administration of appropriate sex hormones may be beneficial.

Linear cauterization of the turbinates may result in some degree of benefit. Removal of turbinates is never advisable as a therapeutic measure. The removal of septal spurs and deflections should be undertaken.

CHRONIC ATROPHIC RHINITIS

Synonyms: Ozena, Primary Atrophic Rhinitis, Sclerotic Rhinitis

Chronic atrophic rhinitis is a disease in which all the tissues of the nose atrophy. The etiology is unknown. The disease is more common in women than in men. It starts usually at puberty but may be present earlier. It is sometimes associated with atrophic vaginitis in girls.

Symptoms and Diagnosis.—The patient complains of crusting in the nose and of anosmia. Examination reveals large, green, usually foul-smelling crusts in the nose. When these are removed the examiner is impressed with the large space in the nose. The turbinates appear as thin shelves jutting from the lateral nasal walls. The membranes are pale. The nasopharynx can be easily seen. *Bacillus ozaenae* (*Klebsiella ozaenae*) and *Perez bacillus* (*Escherichia foetida*) are commonly found in association with this disease. The foul odor of the crusts is said to be due to these bacilli. These organisms, however, are not the cause of the atrophy.

Treatment.—Treatment consists of efforts to keep the nose clean, saline irrigation and oily nasal sprays are helpful. For the fetid odor 25 per cent glucose in glycerin has been found efficacious. It is applied by swabbing in the nose or as tampons. In recent years much has been accomplished in the treatment of atrophic rhinitis by the use of estrogenic compounds as local applications. The nose should be cleansed with an alkaline solution before the estrogenic compound is applied. Variable results have been reported, but most are encouraging. Local treatment with prostigmine methylsulfate, used

to obtain continued local vasodilation, has been reported⁸ and favorable results seen.

Various operations have been suggested for diminishing the size of the nasal cavities, but all have proven ineffective.

SECONDARY ATROPHIC RHINITIS

Secondary atrophic rhinitis occurs in patients with long standing nasal and sinus infection. Mucous membrane changes are noted similar to those seen in primary atrophic rhinitis, but the bony structures of the nose are not atrophied and seldom are there present the fetid crusts which are found in the primary condition. Much thick mucopurulent secretion is present and some crusts, but the odor is usually absent.

Etiology.—This condition is seen always in relation to chronic infection in the nose and paranasal sinuses or as the result of too radical surgery. The drying of the mucous membranes incident to too great space in the nose following ill-advised operation leads to atrophy. The continued discharge of pus in the nose from a chronically infected sinus has the same effect.

Treatment.—Treatment is directed toward curing the chronic infection and the maintenance of a clean nose free from crusts and discharge. To this end, local treatment, such as nasal irrigation, and spraying with vegetable oils, is helpful.

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ALLERGY OF THE NOSE AND PARANASAL SINUSES VASOMOTOR RHINITIS HAY FEVER

Diagnosis of Allergy of Nose and Paranasal Sinuses—Accurate diagnosis of allergy of the nose and paranasal sinuses involves the careful consideration and correlation of the following points

1 *Symptoms*—The typical symptoms of sneezing, itching, nasal discharge, and obstruction are easily recognized. Atypical cases, however, characterized by stuffiness and post-nasal discharge with little or no sneezing, should not be overlooked. Frequent recurring colds, particularly in children, must be differentiated from allergy.

2 *Nasal Changes*—Although the typical allergic mucosa is characterized by pallor, boggy, and sometimes by edema or polyposis, there are many instances in which the mucosa appears normal or somewhat reddened.

3 *Cytology of the Secretions*—Nasal secretions and, when available, sinus secretions should be stained and examined microscopically for eosinophils, neutrophils, or both. The cytologic picture is frequently the most important factor in diagnosis. It must be emphasized that the common cold is an ever present complication, and the cytologic findings are the most important guide in its recognition.

4 *Roentgenograms of the Sinuses*—Varying degrees of cloudiness are the rule rather than the exception in allergic sinuses. Changes may be of short duration and transitory. A cloudy sinus, therefore, does not always indicate infection. Roentgen ray findings should be correlated with the cytologic findings and bacteriologic cultures.

5 *Bacteriologic Examination*—Nasal cultures are not very reliable because of contamination. Sinus secretions for cultures must be obtained under strict aseptic precautions to avoid contamination. The bacteriologic findings should be correlated with the cytologic, and acute, subacute, and chronic complicating infections should be carefully evaluated.

6 *Histopathologic Examination*—All tissues removed from the nose and paranasal sinuses should be examined routinely, and the findings correlated with all the diagnostic factors already enumerated.

Relationship of Otolaryngologic Allergy to Other Manifestations—In addition to hay fever

and perennial allergy of the nose and paranasal sinuses, other associated manifestations in this field should be recognized, such as (1) involvement of the external ear, eustachian tube, middle ear, cochlea, and labyrinth, resulting in deafness, tinnitus, and dizziness, (2) recurring swellings of the parotid and submaxillary glands, (3) involvement of the larynx and esophagus, (4) allergy of the eye, (5) allergic headache.

Other associated manifestations of a local or general nature must also be considered such as (1) bronchial asthma, (2) allergic bronchitis, (3) gastro intestinal allergy, (4) skin allergy—urticaria, eczema, angioneurotic edema, purpura, contact dermatitis, (5) urogenital allergy, (6) serum disease. It is important that the otolaryngologist consider the presence or absence of these manifestations for they point to definite etiologic factors. Respiratory allergy is caused mostly by inhalants such as pollens, dusts, cosmetics, animal danders, and molds. All other manifestations of allergy are predominantly caused by foods and sometimes drugs. In uncomplicated respiratory allergy, food is rarely the primary factor but may not infrequently be a secondary factor. Pure hay fever is usually caused only by pollen, but may be complicated by food sensitivity. If perennial respiratory allergy or other manifestations of allergy are present, the hay fever is definitely complicated, and multiple sensitivities are present.

It is evident that a careful detailed clinical history is important in determining the presence or absence of multiple manifestations and in evaluating all possible etiologic factors. The information derived from skin tests should be correlated with the clinical history and physical findings. The skin tests with pollen are highly reliable, but with other substances they may or may not be significant. Although all skin tests may give negative reactions, the clinical history still remains as an important guide in diagnosis and treatment.

Diagnosis and Treatment of Hay Fever—The pollens which cause hay fever in a given locality must be determined on the basis of field surveys and atmospheric counts. In diagnosis and treatment it is necessary to use only those which are of etiologic significance. For routine diagnosis the cut or scratch test is generally used. For testing the degree of skin sensitivity and for treatment, dilutions of the extracts from 1:100 to 1:10,000,000 are employed. The reactions

noted to serial dilutions are used as a guide in treatment. Intracutaneous tests should be performed, first with the 1 10,000,000, 1 1,000,000 and 1 100,000 dilutions. If a wheal of 15 to 18 mm in diameter is noted in the test made with the 1 100,000 dilution no further tests should be applied. If a somewhat smaller reaction is noted, an additional test with the 1 10,000 dilution may be given. It is not often necessary to test with the 1 1,000 dilution. By following this plan, large skin reactions which may cause an exacerbation of symptoms or constitutional reactions can be avoided.

For treatment I employ the preseasonal and coseasonal methods and have discarded the perennial method. Either intracutaneous, subcutaneous, or combined injections may be given. For almost all tree and grass cases and for about one-half of the ragweed cases I use the intracutaneous method coseasonally, in certain instances giving some injections by either method before the onset of the season. In giving subcutaneous injections by the preseasonal plan small doses at five- to seven day intervals have proved to be most satisfactory and unattended by complicating reactions. By this method an optimum dosage instead of a top dosage is desired. The original doses should start with 0.1 cc of the 1 10,000,000 or 1 1,000,000 dilution according to the degree of skin reactivity and increased by 0.05 cc with each subsequent injection. Top doses should be about 0.1 to 0.3 cc of 1 100,000 in very sensitive patients, 0.1 to 0.3 cc of 1 10,000 in the less sensitive, and 0.1 to 0.3 cc of 1 1,000 in the least sensitive. For intracutaneous therapy 0.02 to 0.05 cc of that dilution which produces a wheal of 18 to 22 mm as determined by the degree of skin sensitivity are used. It is necessary to continue these injections every two to five days during the season according to the symptoms and the atmospheric pollen concentration.

For the most satisfactory results in hay fever treatment, complete skin testing should be performed and particular attention must be directed to the control of complicating sensitivities or manifestations such as those resulting from dusts, molds, and foods.

Treatment of Dust and Food Allergies—In the management of all types of allergy the avoidance or removal of the etiologic factors as determined by the clinical history and skin tests is the ideal plan of approach to the solution of the problem. In the majority of the cases, how-

ever, this is not possible. Therefore, it is often necessary to employ injection therapy, dietary manipulation, or both.

In the majority of the cases of respiratory allergy the inhalants are the most important factors. House dust, occupational dusts, cosmetics, animal danders, and molds are the most important. In most cases I use a composite stock dust and make specific autogenous extracts only in special cases. Good results from dust therapy are dependent upon the establishment of an effective optimum dosage rather than a large top-dosage. The degree of skin sensitivity should be determined by intracutaneous tests as suggested in hay fever cases. The dilutions should range from 1 100,000 to 1 10. The 1 100,000, 1 10,000 and 1 1,000 should be applied first. The 1.100 and 1 10 may be added fifteen to twenty minutes later if indicated. Many patients who have symptoms from dusts show negative skin reactions. Injection treatment should be given in spite of this fact. Injection therapy should be started with the 1 100,000 dilution in cases of marked severity. In extreme cases begin with the 1 1,000,000 and in the mild cases with the 1 10,000 dilution. Injections should be given subcutaneously at five- to seven day intervals increasing each dose by 0.05 cc. When a satisfactory dose is reached the intervals should be increased to ten, fourteen, or twenty one days with no increase in dosage. If the intervals cannot be lengthened, the dose may be further increased as indicated. Optimum doses vary from 0.1 to 0.2 cc of 1 100,000 to 0.1 to 0.3 cc of 1 1,000.

In the management of the food allergies the causative factors may be determined on the basis of the clinical history and the skin tests. If avoidance or elimination of the suspected foods does not result in satisfactory improvement, some type of elimination diet such as suggested by Rowe, Vaughan, and others should be employed. Injection of food extracts has not been consistently satisfactory. Oral hyposensitization has been more effective in restoring tolerance.

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SYPHILIS OF THE NASAL CAVITIES

Syphilis of the internal nose is less frequent than that of the external portion. The skin of the vestibulum nasi may be the site of luetic lesions, but this condition has been discussed under "Syphilis" in the chapter "Dermatoses of the External Nose."

Acquired Syphilis of Nasal Cavities—All three states of luetic infection of the nasal cavities have been observed.

The *primary* lesion (a rare incident) is characterized (1) by intensive redness and swelling of the internal nose and (2) by an indurated chancre, usually on the septum, which is often the site of secondary ulceration. Invariably the mandibular glands are enlarged and hard.

In the *secondary* stage of syphilis, changes, resembling a plain coryza, are usually observed. Some writers claim that a pallor of the middle turbinate, together with hyperemia of other parts, is a characteristic sign. Papular infiltrations are observed only in parts covered by stratified epithelium, whereas Schneider's mem-

brane may show a circumscribed ulceration without a thickening of the epithelium. Extensive ulcerations and necrosis are extremely rare. As a rule, healing occurs without scars.

The *tertiary* stage of syphilis is best known to the rhinologist not because it is a more common type but because its changes are apt to be permanent. Gummas arise from any part of the mucous membrane or skeleton of the nasal cavity, although the paranasal sinuses, especially the frontal sinuses, are sites of predilection. Clinically, there are two types of initial lesion in the tertiary stage, (1) the diffuse infiltration and (2) the circumscribed tumor. Histologically the characteristic lymphatic infiltration is always diffuse. The most frequent locations of tertiary nasal lesions are (1) the septum, (2) the floor, and (3) the roof.

Ordinarily the appearance of the membranous lining is not changed. If the syphilitic process attacks the turbinates, however, a polypoid degeneration may occur. In the later stages of the process the well known atrophy is almost invariably found, an experience which has led some specialists to the erroneous belief that ozena was due to syphilis.

Tertiary syphilis of the skeleton of the nose is conducive to the formation of gummas with rarefaction of bone, but sometimes periosteal thickening of the bone, the so-called "tophus syphiliticus," is observed.

The destruction of bone in tertiary lues causes characteristic nasal deformities. Saddle nose is due to loss of the upper septum and nasal bones. Destruction of the vomer leads to a septal perforation which begins in the bony part, other types of perforation (such as that due to rhinitis sicca, tuberculosis, and fingernail ulceration) are generally found in the cartilaginous portion. A gumma in the floor of the nose may lead to perforation into the oral cavity. In advanced stages of the disease the destruction may be excessive. Seiffert saw a patient in whom the *entire* external nose, maxilla, part of the orbit, all the turbinates, and the hard palate were missing.

Congenital Syphilis of Nasal Cavities—The pathological changes of the nose in congenital syphilis of infants are well known. Clinically and by gross inspection of anatomical specimens, the picture is not characteristic. In addition to the acute rhinitis, that is, the coryza neonatorum, there may be an accumulation of crusts and small excoarations of the skin of the

vestibule In older individuals, gross changes resemble those found in tertiary lues A thorough study of the histologic finding in congenital lues in infants has been done by Hajek and Grossmann They found delay in calcium resorption in the cartilage, increased absorption of bone, absence of osteoblasts, and typical formation of new bone In congenital lues the mucous membrane and the bone marrow show cellular infiltration, epithelial metaplasia, and glandular degeneration In view of these changes, it seems doubtful if complete healing is ever possible

Treatment—Although the diagnosis of syphilis of the nasal cavities is sometimes difficult, adequate treatment does not offer any difficulties General antiluetic treatment, together with mild symptomatic local treatment, has proved adequate in almost all cases In coryza neonatorum the nares have to be kept open to insure adequate ingestion of food

For treatment of systemic syphilis see page 20

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TUBERCULOSIS OF THE NOSE AND NASOPHARYNX

Incidence—Tuberculosis of the nose and nasopharynx appears to be somewhat rare It is symptomless until well advanced, and, in affected individuals, careful examination of the nose and nasopharynx is rarely made until symptoms referable to the nose or throat are present The disease seems to be more prevalent in women than in men and occurs most frequently in persons between the ages of twenty five and fifty years Tubercle bacilli have been found, upon laboratory examination of adenoid tissue, in very young individuals otherwise healthy

Etiology.—Tuberculosis of the nose and nasopharynx may be either primary or secondary

to tuberculosis of the lungs The nasal mucosa and nasal secretion are very resistant to the tubercle bacillus Laboratory experiments show that the nose of healthy individuals may harbor tubercle bacilli Points of lowered resistance in the nasal or nasopharyngeal mucosa, such as those caused by breaks in its continuity due to trauma, predispose to the lodging of the bacilli

Atrophic rhinitis is a predisposing cause Adenoids may be responsible for the development of nasopharyngeal tuberculosis, particularly in children of tuberculous parents In adults with tuberculosis, the nasopharynx is subjected to bombardment by portions of tubercle bacillus laden sputum, particularly in those patients who try to suppress cough Advanced pulmonary and milary tuberculosis frequently are complicated by postnasal and nasopharyngeal tuberculosis, which usually are discovered at autopsy by microscopic examination of nasopharyngeal tissue

Pathology.—Tuberculosis of the nose usually is manifest as an ulcer or tumor mass The infiltrative stage is rarely seen. The tuberculous lesions are most generally seen on the quadrangular cartilage of the septum and more rarely on the inferior turbinate and the posterior nares The floor of the nose is the least common site of the lesions and usually becomes involved by extension from the cartilage of the septum or the turbinate In the nasopharynx, tuberculosis usually is manifest by ulcerations and infiltration of lymphoid tissue A common site is in the region of the fossa of Rosenmuller, and tuberculous otitis media may occur by extension through the eustachian tube In the nose, tuberculosis may extend to the orbit through the lacrimal duct The nasal accessory sinuses and even the meninges may become involved by extension from the nose

Macroscopically and microscopically, tuberculous lesions in the nose and nasopharynx present the same picture as tuberculous lesions in the larynx Tuberculous ulcers of the septum tend to perforate unless arrested by successful treatment

Symptoms—Tuberculosis of the nose and nasopharynx nearly always is secondary to tuberculosis elsewhere in the respiratory system The nasal symptoms are few Usually there is not much annoyance unless there is bleeding upon blowing of the nose Collections of dried secretion adhering to the ulceration on the septum produce a desire to "pick" the nose Tubercu-

lous masses in the nose may be large enough to obstruct nasal breathing or to cause a sensation of stuffiness in the nose

In the nasopharynx, dryness of the throat is a common symptom. The patient has difficulty in locating uncomfortable sensations in the nasopharynx and frequently describes the site as being low down in the throat. The physician should examine the nasopharynx when the patient complains of pain in the throat and no lesion of the oropharynx or larynx can be found, since nasopharyngeal pain may be referred to the throat lower down.

In one case of tuberculous ulcer of the nasopharynx the only symptom was severely painful swallowing. The pain was referred to the larynx, which was found to be entirely free from tuberculosis. Touching the ulcerated area in the nasopharynx with a probe produced pain in the region of the larynx. Healing of the ulcer in the nasopharynx completely abolished the odynophagia.

Diagnosis—The diagnosis is made by careful examination of the nose and nasopharynx, histologic examination of biopsy specimens, examination of nasal secretions for tubercle bacilli, and a history of tuberculosis elsewhere in the respiratory system.

Syphilis and cancer of the nose and nasopharynx must be excluded as they very closely resemble tuberculosis. Tuberculosis usually attacks the anterior cartilaginous portion of the septum, while syphilis usually involves the bony portion near or at the junction of the cartilage, the vomer, and the perpendicular plate of the ethmoid. Tuberculous lesions progress much more slowly and are much paler in color than syphilitic lesions. The ulcers of tuberculosis are shallow with thin, ragged edges while those produced by syphilis are deeper with indurated, elevated, and more sharply defined edges. Sloughing and an offensive odor are likely to be present in syphilis and usually are absent in tuberculosis, unless complicated by chronic atrophic rhinitis with ozena. The Wassermann test is of value in differentiating between syphilis and tuberculosis. Histologic examination of tissue removed from the lesion makes the diagnosis certain.

Prognosis—The prognosis, as in tuberculosis of other parts of the respiratory system, depends mainly upon early diagnosis, before the lesion has become advanced. In early tuberculosis of the nose and nasopharynx, prompt

energetic treatment gives a favorable prognosis. Untreated ulcer of the septum may progress to perforation. When untreated, the lesion is slowly progressive and shows little tendency to heal. Recurrence is frequent. Extension to the sinuses, orbit, and meninges presents a grave prognosis.

Treatment—A very important part of the treatment is keeping the nose clean and clear of secretions and crusts. The extent and vigor of the treatment depend upon whether the lesion is circumscribed or extensive and whether or not it is secondary to pulmonary tuberculosis.

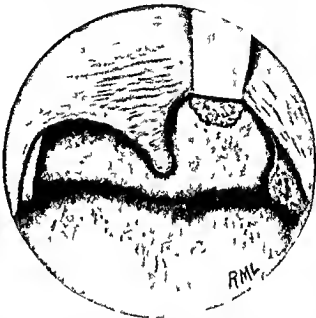


Fig 19—Tuberculous ulcer of the nasopharynx. This patient complained of severe dysphagia. Examination of the larynx showed no abnormal condition. The ulcer, which was discovered by routine examination of the nasopharynx, healed promptly as a result of 10 per cent silver nitrate applications. Dysphagia disappeared after the first treatment.

Alkaline antiseptic irrigations should be used to clear the nose of secretions. Crusts should be softened and removed before making applications to the ulcers. The ulcerations should be treated conservatively if they show a tendency to heal. Applications of chaulmoogra oil, full strength, have proved very useful. Lactic acid, chromic acid, trichloroacetic acid, and other cauterants have been advocated in the past, but I have had very little success with them. In applying the cauterants, care should be taken to avoid damaging surrounding mucosa by the use of a sloppy swab. Personally, I prefer to use chaulmoogra oil applications, when more radical treatment is not indicated.

The electrocautery is of great value in treating these ulcers, if care is taken to avoid accidents caused by careless handling of the instrument. The technic used is the same as that used in treating the larynx.

If the above methods are not effective, more radical treatment is indicated. The ulcer may be excised, removing all of the tuberculous tissue, the resulting raw surface is then treated as a simple ulcer. Tuberculous tumors and granulomatous masses should be excised and the site treated as described for the tuberculous ulcer.

When advancing tuberculosis of the lungs exists and the general prognosis is bad, only conservative treatment should be used as a palliative measure. When the nasal and nasopharyngeal lesions are a part of an advanced progressing pulmonary tuberculosis or a miliary tuberculosis, no extensive or radical treatment should be undertaken. In all cases the patient should be under the supervision of the chest physician, and the nature of the rhinologic care governed by the condition of the lungs and that of the patient in general.

ROBERT M. LUKENS

DISEASES OF THE NASAL SEPTUM

Deviations of the Nasal Septum.—Etiology.—The exact cause of the various types of deviations or malformations of the nasal septum is unknown. The great majority seem to be developmental rather than traumatic. They are rare in young children but quite common in adults. In fact, the adult who is entirely free from some deviation or abnormality of the septum is the exception. Deformities of the septum are seldom observed in the Negro.

Types of Deviations.—The septal abnormalities may be either bony or cartilaginous. They may take the form of bends, ridges, spurs, or any combination of these.

Anterior deviations are usually cartilaginous, either involving the columnar cartilage in which the antero-inferior border of the cartilage is turned outward into the vestibule of the nose, partially obstructing the respiratory passage, or a bending or curvature of the quadrilateral cartilage, either in an anteroposterior, a perpendicular, or a combination of directions.

Osseous deviations frequently take the form of a bony ridge at the upper border of the nasal crest of the maxilla or the superior border of the vomer. These ridges are directed, as a rule, in an upward and backward direction. Impaired respiration may be the result of this type of septal deviation. The perpendicular plate of the ethmoid is frequently to one or the other side in a convex or cup-shaped deformity. This type of deviation may impinge upon the middle turbinate on the side of convexity, thereby interfering, to some extent at least, with the drainage and ventilation of the sinuses.

Isolated deformities, such as spurs or short ridges, may be observed. These spurs may be composed of either bone or cartilage. They are located, as a rule, at the junction of the anterior tip of the vomer and the quadrilateral cartilage and, to a lesser degree, at the junction of the quadrilateral cartilage and perpendicular plate of the ethmoid. Symptoms are usually absent from this type of deformity.

Symptoms.—Symptoms, if any are present, are those of impaired nasal respiration from the inferior type of deviation or ridge or from interference with the ventilation or drainage of the sinuses from deviations in the upper portion of the septum.

Indications for Surgical Correction.—If the malformation of the septum interferes with proper nasal respiration on either side, or with the drainage or ventilation of any of the sinuses, or impinges upon any of the turbinates interfering with their function, the malformation should be corrected by the proper surgical procedure.

Contraindications are advanced tuberculosis, untreated syphilis or diabetes, atrophic rhinitis, acute respiratory infections, marked hyperplastic rhinitis, and incomplete facial development in the case of children.

The Submucous Resection of the Septum.—ANESTHESIA.—The patient is placed in a sitting or semireclining position in a chair, or on an operating table with a movable back and head rest—if the patient feels faint at any time during the operation, the tilting back permits a horizontal position to be assumed.

The preoperative administration of an opiate is optional. Local anesthesia is obtained by swabbing the septal mucous membrane with two or three applications of a 10 per cent cocaine solution or cocaine flakes. If the latter is used, a cotton-tipped applicator is moistened

with 1,000 epinephrine solution the excess moisture squeezed out and the moistened tip inserted into the cocaine flakes. This is then gently massaged over the entire surface of the nasal septum. Two or three applications are usually sufficient. If it is necessary to resect the columnar cartilage a 1 per cent or 2 per cent procaine solution with 12 drops of epinephrine hydrochloride to the ounce is injected subcutaneously and submucously over the portion to be resected.

THE INCISION—The curved Killian incision is usually used (Fig 20). It is made anterior to

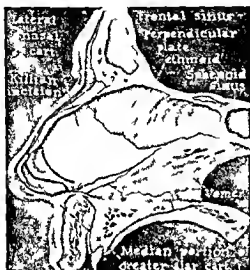


Fig 20—The Killian incision. The septum is shown denuded of its membranous covering. The label Lateral refers to quadrilateral.

is inserted in an upward and backward direction beneath the bridge of the nose, and the elevator is then swept downward (Fig 21) until the vo-

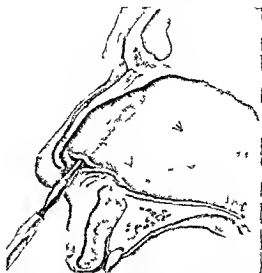


Fig 21—The semioval elevator has been introduced beneath the mucoperichondrium in an upward and backward direction. The elevator is then swept downward and backward into the upper crest of the vomer elevating the mucoperichondrium with the shank of the instrument. The flat side of the elevator is in contact with the septal cartilage.

mer is reached. At times a sharp elevator is necessary to elevate the periosteum (Fig 22).

INCISION THROUGH THE CARTILAGE—This is accomplished by means of a small, short

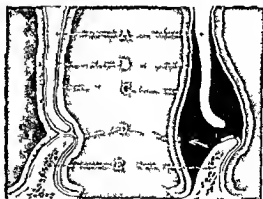


Fig 22—Sections through the nasal septum before and after elevation of the soft structures. A quadrilateral cartilage, B perichondrium, C, mucous membrane, D periosteum, E vomer. The periosteum has been incised along the crest of the vomer and elevated on one side.

bladed sharp knife, great care being taken to avoid cutting through the opposite mucoperichondrium. If this should occur and one or the other side is not sutured, a permanent perforation will result. The tip of the left index

the deviation and posterior to the junction of the vestibular and mucous membranes of the septum where the perichondrium elevates with ease. The incision is made with a small sharp pointed knife. Care should be taken to avoid penetrating through the cartilage into the opposite mucoperichondrial membrane. This mishap may be avoided by inserting the tip of the left index finger into the opposite nostril for counter pressure and guidance while making the incision.

ELEVATION OF THE MUCOPERICHONDRUM AND PERIOSTEUM—Elevation of the mucoperichondrium is begun by teasing the membrane away from the cartilage by means of a flat sided semisharp elevator. Care should be taken to avoid separating the mucous membrane from the perichondrium. This can be determined by the white, glistening appearance of the cartilage when properly elevated. After the elevation has been started a blunt semislat rounded elevator

Treatment—Crusting should be reduced to a minimum by local cleansing washes and ointments. The application of the electric cautery to recurrent bleeding points may be necessary.

If the perforation is small a plastic flap of mucoperichondrial membrane should be separated from an adjacent portion of the septum and turned over the opening and sutured beneath the elevated mucoperichondrium around the perforation. If the perforation is so large that a unilateral flap will not cover the opening a plastic flap on the opposite side may cover the remaining portion.

In small or medium sized perforations the edges of the hole are freshened and the mucoperichondrium elevated anterior to the perforation on one side and posterior to the perforation on the opposite side. A long curved incision is then made $\frac{1}{2}$ to $\frac{3}{4}$ inch from the perforation where the elevations have been made on each side of the septum. The mobilized ribbon flaps are then brought over the perforation and sutured to the opposing mucoperichondrium on each side (Fig. 24). The sutures are removed in from one to three days.

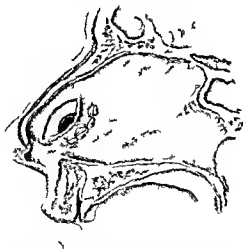


Fig. 24—Plastic septal flap for closing a small perforation of the septum. A similar flap is made on the opposite side to cover any remaining portion of the perforation not closed by the first flap.

In large perforations it is impossible to close the opening.

Epistaxis (Nasal Hemorrhage)—Etiology—The great majority of nosebleeds are from varicose veins in Kiesselbach's area or arteries located in the antero-inferior portion (Fig. 25) of the cartilaginous septum (Little's area).

Bleeding from the nose frequently accompanies acute infections of the nose, trauma, foreign bodies in the nose, violent exertion, high altitude, septal ulcers, chemical intoxication such as mercurial and phosphorus poisoning, angiomatous or malignant growths of the nose, vicarious menstruation, and many constitutional diseases.

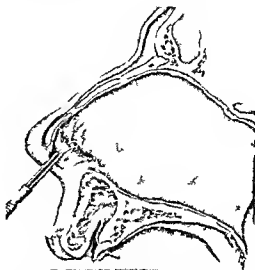


Fig. 25—Cauterizing the veins (Kiesselbach's plexus in Little's area) for frequent nosebleeds.

such as hemophilia, purpura, leukemia, syphilis, tuberculosis, certain iron deficiency anemias, vitamin C deficiency, and hypertension.

Treatment—The treatment of nosebleeds is directed to the cause as well as to the immediate stoppage of the bleeding. If bleeding is from the anterior portion of the septum a small pledget of cotton placed within the anterior nares and left in place for from a few minutes to two or three hours will check most nosebleeds. Moistening the cotton pledget with 1:1000 epinephrine solution or a 1 per cent to 3 per cent epinephrine hydrochloride solution will help control the bleeding. A semirecumbent position and physical relaxation reduce the tendency to bleed.

If the bleeding has been severe or has occurred repeatedly, destroying the enlarged blood vessels with the electric cautery is preferable. A blunt pointed electrode is heated to a cherry red heat and inserted for a moment into the bleeding area. Two or three applications to various areas may be necessary. The preliminary application of cocaine is advisable.

If bleeding is intractable and the bleeding points cannot be determined, packing the affected nostril with $\frac{1}{4}$ -inch selvedge gauze

finger in the nostril opposite the incision helps prevent this mishap. The incision through the cartilage should be large enough to permit the insertion and manipulation of the flat-sided, semisharp elevator through the opening.

ELEVATION OF THE OPPOSITE MUCOPERICHRONDRUM AND PERIOSTEUM—The elevator is inserted through the incision in the cartilage with its flat side in apposition with the cartilage. The elevator is inserted in an upward and backward direction along the bridge of the nose and then swept downward as described for the opposite side. The area elevated on each side should be considerably larger than the deviated portion to be removed. If a sharp, bony ridge is present

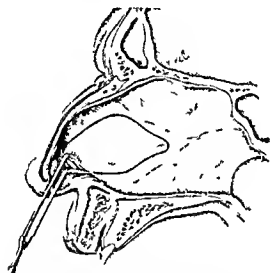


Fig. 23—The Ballenger swivel knife is introduced through the mucoperichondrial incision. The swivel blade is placed astride the cartilage and the deflected portion encircled and removed.

along the edge of the vomer, careful dissection with a sharp or semisharp, curved elevator may be necessary.

REMOVAL OF THE CARILAGINOUS SEPTUM—This is readily accomplished by means of the Ballenger swivel knife. The swivel blade is placed astride the upper portion of the incised cartilage (Fig. 23) and then pushed in an upward and backward direction until the bony septum is reached. It is then swept downward to the vomer and then drawn forward along the upper border of the vomer to the inferior portion of the incision. The incised cartilage is then grasped with a dressing forceps and removed through the incision.

REMOVAL OF THE PERPENDICULAR PLATE OF THE ETHMOID—Cutting bone forceps of the

Foster-Ballenger or other type is inserted into the mucoperichondrial pouch, and by taking successive bites all deviated portions are gradually removed. Care should be taken to avoid excessive twisting motions while removing this plate of bone as the cribriform plate might possibly be fractured.

REMOVAL OF THE VOMER—In most instances it is necessary to separate the inferior attachment of the vomer to the nasal crest by means of a V-shaped gouge and hammer. When the vomer is loosened, it may be grasped with heavy forceps, and by a gentle twisting or rocking motion the posterior attachment will separate. Care should be taken at this stage to avoid tearing the two membranes opposite each other as a permanent perforation might result.

THE DRESSING—After all the septal abnormalities have been corrected and the flaps coapted, a $\frac{1}{2}$ inch, selva edged gauze tape soaked in petroleum jelly is carefully packed into each nostril so as to hold the septal flaps in the midline position. The dressing is removed in twenty four hours, and if bleeding occurs or the flaps do not stay in position, the dressing may be reinserted on one or both sides for another twenty four hours. If crusts tend to form over the nasal mucosa, oil or petroleum jelly may be applied to the involved area. Dusting the nasal cavity with one of the sulfonamide powders controls any postoperative infection.

Perforation of the Nasal Septum—*Etology.*—Perforation of the septum may be congenital or acquired. The congenital type is very rare. The acquired type may be due to trauma (usually operative), chronic granulomas, such as syphilis, tuberculosis, or lupus, or to acute or chronic infections with ulcerations, such as are occasionally seen with typhoid, diphtheria, scarlet fever, and abscesses. An atrophic type of ulceration of the cartilaginous septum may eventually result in a perforation.

The syphilitic perforation has a predilection for the junction of the cartilaginous and bony septum. The tuberculous and traumatic perforations usually involve the cartilaginous septum.

Symptoms—Symptoms are frequently absent. If the perforation is small, a whistling sound may be present on either inspiration or expiration. In the larger perforations, crusting with or without partial nasal obstruction may be present. If the crusts adhere to the nasal mucosa, slight nasal bleeding is noted when the crusts are removed or blown out.

Treatment—Crusting should be reduced to a minimum by local cleansing washes and ointments. The application of the electric cautery to recurrent bleeding points may be necessary.

If the perforation is small, a plastic flap of mucoperichondrial membrane should be separated from an adjacent portion of the septum and turned over the opening and sutured beneath the elevated mucoperichondrium around the perforation. If the perforation is so large that a unilateral flap will not cover the opening, a plastic flap on the opposite side may cover the remaining portion.

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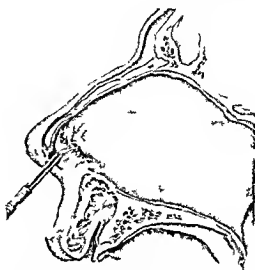


Fig. 25—Cauterizing the veins (Kiesselbach's plexus in Little's area) for frequent nosebleeds.

such as hemophilia, purpura, leukemia, syphilis, tuberculosis, certain iron deficiency anemias, vitamin C deficiency, and hypertension.

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If bleeding is intractable and the bleeding points cannot be determined, packing the affected nostril with $\frac{1}{2}$ inch selvedge edged gauze

tape soaked in petroleum jelly is necessary. The gauze is first inserted into the upper posterior portion of the nasal cavity and then packed forward and downward until the packing has been completed. The packing should be removed in twenty-four hours but may be reinserted if necessary.

In severe nasal hemorrhage, when other means have failed, blood transfusions are usually successful. In rare instances ligating the external carotid is essential. The internal carotid artery should be ligated if the bleeding is from the upper part of the nose as that portion is supplied by the anterior and posterior ethmoid branches of the ophthalmic artery, a branch of the internal carotid.

Hematoma of the Septum—*Etiology*—A collection of blood beneath the perichondrium or periosteum of the nasal septum is usually due to trauma, but may occur in the various bleeding diseases. It frequently follows the carefully performed submucous resections in which tears are absent.

Symptoms—Partial or complete nasal stenosis is the most prominent symptom. This is usually bilateral as the hematoma generally involves both sides. Headache of varying severity is frequently present due to pressure of the swelling within the nose.

Diagnosis—The diagnosis is made by inspection and palpation. If doubt exists as to the nature of the fluid within the septum, a small diagnostic puncture may be done.

Treatment—A free incision near the floor of the nose, on both sides if necessary, should be made. It may be advisable to keep the incision open for a few days or until the bleeding has stopped. A light packing to hold the distended pouch together may be necessary at times.

If left to itself without drainage, the blood clot may result, in time, in a greatly thickened septum with much fibrous tissue.

Abscess of the Septum—*Etiology*—An abscess of the septum is usually due to trauma in which a hematoma forms with a secondary infection of the clot. Occasionally an abscess follows an acute infection such as typhoid, scarlet fever, or measles.

Symptoms—The abscess may require several days to develop to its full extent. After pus has formed, marked swelling, redness, and edema on one or both sides of the septum are observed. The inflammation and edema may extend to the external nose. Destruction of the cartilage may

occur, but, as a rule, the mucoperichondrium remains intact.

Treatment—Free incision of the abscess at its most dependent portion should be made as soon as fluctuation is apparent. A gauze drain may be necessary for a few days to maintain an open incision. One of the sulfonamide powders dusted into the abscess cavity shortens the healing time.

Rhinitis Sicca Anterior—Dry anterior rhinitis is an atrophic change in the mucous membrane of the antero-inferior portion of the nasal septum. It is characterized by a localized change in the mucous membrane from the normal pseudociliated columnar epithelium to a flat, squamous type with an impaired glandular function. The involved area appears as a whitish mucous membrane covered by a thin, dry, pseudomembrane with dry crusts somewhat adherent to the underlying mucosa. Repeated forceful removal of the crusts or dry membrane perpetuates the condition so that in time superficial dry ulcers may form. Slight bleeding from the involved area is common. The ulceration, if deep or long continued, may perforate the cartilaginous septum.

Treatment—Picking the nose or forceful removal of the crust or pseudomembrane should be avoided. Any bleeding points should be cauterized with a small-tipped electric cautery. A bland ointment will lessen the tendency to crust formation.

Atresia of the Posterior Choanae—Choanal atresia or imperforation of the posterior choanae is rare. It may be congenital or acquired, unilateral or bilateral, complete or incomplete, bony or membranous, or a combination of these various types.

Etiology—Atresia of the posterior choanae is usually congenital, due in most instances to the persistence of the nasohyaline membrane. The acquired type, usually located in the pharyngeal region (considered elsewhere) rather than in the posterior choanae, is due to such causes as trauma or severe infections (syphilis, diphtheria, and tuberculosis, for example) in the nose or posterior nares with a subsequent cicatrix formation closing the posterior choanae or nasopharynx.

Pathology—The congenital type of posterior choanal atresia, either membranous or osseous, is usually located near the posterior end of the vomer. In adults the unilateral type is seen more frequently than the bilateral type. In infants the

bilateral type is found more frequently. The mucous membrane and turbinates may, on the affected side and after several years, show some hyperplasia or even atrophy.

Symptoms and Diagnosis—The predominant symptom is the absence or impairment of breathing on the affected (or both) sides. If bilateral in an infant, difficulty in nursing is observed from the first day. The nasal cavities are filled with a glairy mucus which does not drain well. Anosmia is present on the affected side.

Examination with a probe through the nasal passages or with a nasopharyngoscope or palpating finger in the nasopharynx reveals the condition.

Treatment—In infants, if the occlusion is membranous, spontaneous rupture may occur, with relief of the symptoms. In most instances, however, the surgical removal of the obstruction is required. The partition is removed entirely, including a portion of the posterior septum. Care should be taken to avoid injuring the pterygopalatine canals situated immediately anterior to the choanal orifices.

HOWARD CHARLES BALLENGER

MAXILLARY SINUSITIS

Acute Maxillary Sinusitis—Acute maxillary sinusitis (acute inflammation of the antrum of Highmore, acute antritis) is an acute inflammatory process of the mucous membrane which lines this cavity.

Etiology—This condition is due to an extension of an infection from the nasal cavity through the maxillary ostium or an accessory opening (present in 25 per cent of cases) or through the floor of the sinus from a tooth-root infection or it may be due to a transmission of organisms through a break in the floor following the removal of an upper molar or premolar tooth. Predisposing factors may be anatomic or pathologic and include all factors which interfere with sinus drainage and ventilation. A blocked middle meatus is commonly the cause of impaired sinus function. This may be due to pressure on the middle turbinate by a septal deviation or spur, or to the pressure of a large cellular turbinate (concha bullosa). From a pathologic standpoint, predisposing factors are

the presence of polyps or other new growths in the middle meatus, edema of the tissues associated with allergy, or an inflammatory process of an acute or chronic nature in one of the other sinuses of the anterior group, the ethmoid or frontal.

Pathology—The maxillary sinus is lined with pseudostratified ciliated columnar epithelium which is continuous with that of the nasal cavities. As is usual with respiratory epithelium a clear-cut basal cell layer is present. The sinus mucosa, however, differs from that of the nasal cavity in that there is a preponderance of goblet cells. The few glands present are located near the ostium. The ciliated mucosa acts as a primary defense mechanism against invading organisms. Covered with a mucous film, the cilia sweep in the direction of the sinus outlet. If there is no impairment to drainage the mucus, laden with foreign material, passes into the nasal cavity and thence to the nasopharynx.

Acute inflammation consists, in the early stages, of hyperemia and exudation through the dilated capillary walls. The exudate consists of serum, fibrin, and polymorphonuclear leukocytes. Within a few hours after the onset lymphocytes and plasma cells are added and, subsequently, histiocytes. The early edema characteristic of acute inflammation soon begins to subside but cellular infiltration and proliferation continue. The surface cells which early are lost are replaced by cells from the basal layer. As the edema subsides the sinus ostium becomes patent and the exudate drains into the nasal cavity.

Symptoms—The extent of the symptoms varies with the virulence of the organisms and the local and general resistance of the host. There may be a fever, prostration, and a feeling of severe illness, or the patient may feel well enough to carry on his duties as usual. This is more likely to be the condition found in recurring cases. The early stage is characterized by pain and tenderness over the cheek, nasal voice, and discharge. Pain may be felt over the eye or in the teeth on the involved side. Examination of the nose may reveal pus in the middle meatus. The turbinates are usually red and edematous. There may be a postnasal discharge which can be observed on the pharyngeal wall or by mirror examination of the nasopharynx.

As the disease progresses, the drainage improves and the pain diminishes with the increase

of the discharge. Symptoms may then focus in the throat which may be irritated and there may be an annoying cough.

Diagnosis—To aid diagnosis rhinoscopy (anterior and posterior), transillumination, roentgen-ray studies, and diagnostic lavage are available. In most cases the history of a recent head cold simplifies the diagnosis. If the patient is vague about the onset of his symptoms, sinus disease of dental origin should be considered. Dentogenic infections of the antrum occur in approximately 10 per cent of all cases and are characterized by the presence of a foul-smelling nasal discharge.

A similar discharge is significant of a foreign body in the nose and this may be considered if careful search fails to reveal a sinus infection. Disease of the anterior ethmoid cells and frontal sinus may also be confused with a maxillary sinus infection. In some cases the exact location of the infection can only be determined by sinus lavage, and in certain instances even this method is inadequate.

Complications—An antral infection may extend to cause an involvement of other sinuses, it may cause a closure of the eustachian tube, or extend by way of the tube to the middle ear and thence to the mastoid, or the infection may extend to the pharynx and lower respiratory tract.

Intracranial complications are rarely traced to the maxillary sinus, yet, in exaggerated cases with blocked drainage, there may be an extension of the process through the bony walls to the orbit, cheek, palate, and alveolar process. These cases are rare, as are those of osteomyelitis unassociated with surgical intervention.

Sequelae—Acute attacks of maxillary sinusitis may clear up spontaneously, terminate with the aid of proper therapeutic management, or persist as a subacute or chronic type of infection.

Treatment—**PROPHYLAXIS**—Acute attacks of maxillary sinusitis may be eliminated completely in many cases if, between attacks, measures are instituted to improve the sinus drainage space. This includes corrections of all abnormalities, removal of growths, control of allergy, and a hygienic regimen which will lessen the tendency to nasal infections.

MEDICAL CARE AND MANAGEMENT—In the early stages, very little treatment is indicated or of value. It is generally conceded that acute sinusitis has a marked tendency to subside spontaneously and that those cases which persist do

so because of drainage impairment. Patients are put to bed when possible in a warm, well-moistened room and efforts are confined to the relief of symptoms and prevention of complications. Pain is relieved by sedatives and heat, moist or dry over the affected area, and nasal blockage is lessened by the instillation of carefully selected nose drops. The ideal preparation is a normal saline solution containing 1 per cent ephedrine. These drops are best applied with the patient in the position of Proetz with the head low, or the side posture of Parkinson. Synthetic preparations such as neosynephrin hydrochloride (0.25 per cent) and privityne (0.1 per cent) may be substituted for the ephedrine.

SURGICAL PROCEDURES—**Infracture of Middle Turbinate**—This procedure is indicated when the middle turbinate is crowded against the lateral nasal wall so as to interfere with sinus drainage. It is contraindicated in the presence of acute congestion of the nasal tissues. A flat-bladed instrument, such as the handle of a thin scalpel or a heavy mucous membrane elevator, is the only instrument required.

Cotton saturated with 5 to 10 per cent cocaine solution is inserted into the middle meatus and left in place for ten minutes. The instrument is inserted into the slit between the middle turbinate and the lateral wall and firm pressure against the turbinate forces it toward the septum.

IRRIGATION OF THE SINUS—This procedure is indicated when there is a persistence of purulent exudate in the sinus. Sinus lavage is *contraindicated* during the early stages before local immunity has been established.

Antral cannulas with dull tips are used in the natural ostium, while sharp-pointed cannulas are used to penetrate the membranous wall of the middle meatus. The curved Coakley trochar or straight Lichwitz needle with Wolf attachment is used for puncture through the osseous naso-antral wall.

In the *inferior meatal approach* the anterior one third of the inferior meatus is anesthetized, cotton containing a 5 or 10 per cent cocaine solution, or some equally effective preparation such as pontocaine (2 per cent solution), is placed in the meatus. The tip of the cannula is inserted high in the vault of the meatus and forced through the wall. The patient's head is then bent forward and the sinus irrigated with a warm normal saline solution. As the sinus fills

with solution, purulent material rises to the top and passes through the ostium to the nasal cavity, thence to a receptacle held beneath the patient's chin.

The tip of the cannula should be well within the sinus lumen before irrigation is begun. It may pass through the roof of the orbit or the lateral wall, or lodge within the sinus mucosa, and injection of fluid or air may result. Such mishaps may be of a minor nature yet air bubbles in the tissues may produce embolism which may be a serious complication. Cases of osteomyelitis following puncture of the naso antral

the bulla and the uncinate process. As it comes into the hiatus semilunaris further rotation brings the tip downward and laterally to pass into the ostium. The opening may be reached by this technic in 55 per cent of cases. In the others the anatomic relations are such as to render the ostium completely inaccessible or to be reached only with difficulty. In the 45 per cent not easily available the membranous wall is punctured with a sharp pointed cannula. Whether the ostium or an accessory ostium is reached, or an opening made in the wall, the irrigation is carried out as described above.

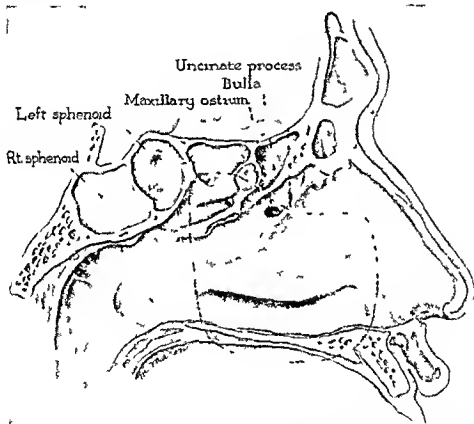


Fig. 26—The maxillary ostium shown in this specimen is easily accessible because of a low, short uncinate process.

wall have been reported but are exceedingly rare.

In the *middle meatal approach* the area is anesthetized by pledgets of cotton containing 5 to 10 per cent cocaine solution placed in the middle meatus. The cannula is placed in the nose above the inferior turbinate with the tip pointing upward to a point halfway back in the nasal cavity. Rotation laterally brings the tip in contact with the lateral wall of the middle meatus and the area is probed for an accessory opening (present in 25 per cent of all cases). The cannula is brought forward and the tip passed between

Prognosis—If proper treatment is not carried out, cases of acute maxillary sinusitis may persist in a subacute stage and the membranes undergo changes significant of chronic infection.

Subacute Maxillary Sinusitis—This condition is a prolongation of an acute infection and treatment is similar to that in the acute infection. If after a few weeks there is no improvement it may be assumed that drainage from the sinus is inadequate. These cases usually clear up following intranasal antrostomy in the inferior meatal wall.

Intranasal Antrostomy (Antral Window).—Instruments needed for this operation are a heavy flat-bladed instrument for elevation of the inferior turbinate, Wagner biting-forceps, which will cut in an anterior, posterior, and downward direction, and a Myles, or similar antrum chisel.

Following anesthesia of the inferior meatus, pressure is applied beneath the inferior turbinate and it is forced upward. Some authors precede this technic by severing the anterior attachment of the turbinate to the nasal wall. An opening is made in the sinus wall of the inferior

Some authors advocate no postoperative packing, others insert 5 per cent iodoform gauze which is tightly packed and left in place for three or four days or even longer.

Care should be taken to *avoid* the nasal orifice of the nasolacrimal duct which is located in the upper anterior portion of the inferior meatus.

POSTOPERATIVE COMPLICATIONS.—Osteomyelitis has been reported to have developed following a window resection. These cases are rare and are thought to result from indiscriminate use of a rasp on cancellous bone.

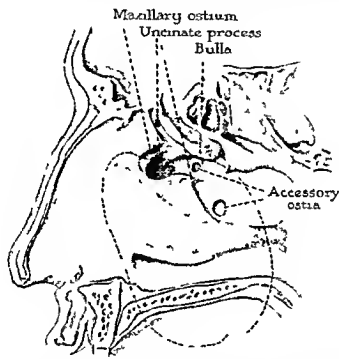


Fig. 27.—In this specimen the maxillary ostium is large yet inaccessible for probing because of a high uncinate wall (13 mm). Irrigation could be carried out through an accessory ostium.

meatus with the antral chisel, and this is enlarged in all directions by means of the cutting forceps. Special attention should be given to the naso-antral ridge which is lowered by bites from the cutting instrument. The bony wall is thickest at this point but the success or failure of the procedure depends in large measure on the manner in which this step of the operation is carried out. The opening in the sinus should be large enough to afford an inspection of its contents and examination of the mucosal lining. If polyps are present and within reach they are removed with grasping forceps or snare and if a cyst is located its walls may be broken down by means of the antral chisel or a curved rasp.

Recurrent Maxillary Sinusitis.—In some individuals attacks of acute antritis develop with nearly every head cold. Between attacks measures should be introduced as indicated to remove drainage barriers. Proper management of these cases may require (1) allergy control, (2) removal of growths in the nose and nasopharynx such as polyps and adenoids, (3) correction of deformities such as septal spurs and deviations, and blocking middle turbinates, (4) intranasal antrostomy.

Chronic Maxillary Sinusitis (Chronic Antritis).—Etiology.—The exciting factors are (1) repeated attacks of acute maxillary sinusitis, or a single attack which persisted to a chronic state,

(2) a neglected or overlooked dental focus. Predisposing factors are drainage barriers such as those enumerated above as being responsible for recurrent attacks.

Pathology—The fundamental pathologic change in chronic inflammation is that of cellular proliferation. Histocytes and fibroblasts predominate, with frequently an admixture of lymphocytes and plasma cells. The exudate with this type of cell proliferation, and in the absence of polymorphonuclear cells, is of a

have as a basis this same process of cellular proliferation.

Symptoms—The systemic effects are those of a low-grade toxemia. Cases are reported of the antrum as an infective focus, yet these are not common. Nasal discharge of a purulent or mucopurulent nature is the expected finding. Postnasal discharge is present and this may be associated with irritated throat, cough, and huskiness of voice. The ear may be affected by tubal blockage. There is seldom headache or

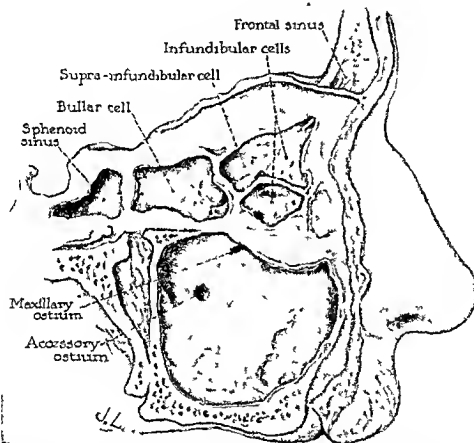


Fig 28—The maxillary sinus and its relations to the other sinuses. The ethmoids and sphenoid have been exposed through the orbit and the lateral wall of the antrum has been removed. Note the thickened, irregular mucosa indicative of chronic infection which lines all of the cavities.

puriform nature and the condition may be designated as a subchronic inflammation. In a more active state (subacute exacerbation) polymorphonuclear cells are added and the discharge becomes purulent. The process of cellular proliferation involves the stroma chiefly producing a granulation tissue with a thickening of varying degree. All variations of chronic sinusitis, whether the clinical picture is that of fibrosis, villous hypertrophy, cystic degeneration, or metaplasia of the epithelium,

fever except in an active subacute exacerbation or in cases of complete blockage to sinus drainage. Pus may be observed in the nasal cavity, middle nasal meatus, postnasal space, or pharynx.

Diagnosis—The presence of pus in the middle meatus although diagnostic of antral disease is also significant of anterior ethmoid and frontal sinus infection, these must be ruled out before the diagnosis of antral infection is established on the basis of pus alone. Transil

lumination, roentgen ray studies with or without opaque media, and diagnostic lavage should, however, establish the diagnosis fairly well.

Complications—Intra orbital, otitic, and pulmonary affections are often traced to chronic maxillary sinusitis. Intracranial complications are rare but may follow a frontal sinus infection which was itself due to an extension of a maxillary sinus infection.

Sequelae—Chronic inflammation frequently ends successfully, the result being characterized by fibrosis. In other cases a subchronic type of infection may result with a nonpurulent exudate present. This exudate is composed of mucus and nonpurulent inflammatory plasma cells and lymphocytes. Other sinuses continue as sup-

nate should be attempted in the chronic cases before radical measures are indicated. These, as cited above, consist of removal of drainage barriers and irrigation therapy. Should these measures fail to effect improvement in symptoms, window resection is advocated. This together with allergy control and proper hygienic care should effect a cure in most cases.

Should disagreeable symptoms still continue a radical removal of sinus mucosa should be considered. This should only be done, however, after a careful survey reveals the fact that all factors which may possibly contribute to the lingering infection have been eliminated. This exhaustive search is advocated with the idea in mind that radical removal of sinus mucosa is



Fig. 29—Filling defect of right maxillary sinus caused by polyp

purating cavities indefinitely, the lining mucosa of these sinuses is thickened and the exudate is of a purulent nature.

Treatment—**PROPHYLAXIS**—Careful attention directed to the acute attack with removal of drainage barriers between attacks and proper dental care should prevent extension of the disease to a chronic state.

MEDICAL CARE AND MANAGEMENT—Medical treatment is of little value in chronic antral disease. Physical measures such as roentgen ray therapy and short wave diathermy are advocated but are of questionable value without the aid of surgical procedures.

INDICATIONS AND CONTRAINDICATIONS FOR OPERATIONS—Surgical procedures of a minor

not an assurance of a cure of the disease and that in very few instances is the procedure indicated. The membrane to be removed, although greatly thickened, polypoid and irregular, may not necessarily be diseased, but with an intact epithelium and a stroma containing powerful inflammatory cells is a defense process, it is resisting disease and capable of doing so indefinitely.

RADICAL SURGICAL PROCEDURES—Two techniques are available, the Caldwell-Luc, the one in common use, and the Denker. The necessary instruments and materials are retractors, a scalpel, hemostats, a periosteal elevator, chisels, bone-cutting forceps, curets, iodoform gauze, and suture material.

In the *Caldwell Luc method* the operation may, in most instances, be carried out under local anesthetic. Cotton containing cocaine solution is placed above and below the inferior turbinate. Novocaine (1 per cent) or a similar solution is injected deeply under the periosteum of the canine fossa. The upper lip is elevated and held up with a retractor. An incision down to the bone is made along the gingivolabial fold between the canine and the second molar teeth. The periosteum is elevated up to the infra-orbital canal, care being taken to avoid injuring the nerve. An opening is gouged into the facial wall of the antrum and this is enlarged by means of the bone cutting forceps to a size which permits inspection of the cavity. Radical removal of the entire sinus mucosa is rarely required, but if this is deemed advisable it is accomplished by means of an elevator or curet. In the event that a window resection had not been done previously this is then performed (For technic see 'Intranasal Antrostomy'). If a mucous membrane flap is available it is turned into the antral cavity and rests on the floor. The turbinate need not be disturbed although some technics call for removal of its anterior portion. Some clinicians insert iodoform gauze which is removed after three or four days. The sublabial wound is closed by interrupted sutures.

The *Denker operation* brings the anterior margin of the opening close to the midline and permits better inspection of the anterior of the cavity both during and after the operation.

There are certain *dangers* to be avoided in these operations. Disturbances of the lacrimal passages may be caused by curet or forceps. Anesthesia of the cheek and teeth may follow injury to the infra-orbital nerve or nerves of the teeth during chiseling of the bony wall.

Complications—Swelling of the cheek is common following the sublabial approach. This usually disappears within a day or two. There may develop an abscess or permanent fistulas, as well as osteomyelitis, which may prove fatal.

Prognosis—Aside from those cases in which osteomyelitis develops, fatalities resulting from the operation are few. The membrane which replaces that which was removed is fibrotic and less disease resisting. The cavity is not obliterated and is always liable to reinfection.

FRONTAL SINUSITIS

Anatomy of the Frontal Sinus—The frontal sinuses are located between the outer and inner tables of the frontal bone. They vary in size, shape, type, and number and may be asymmetrical. Usually they are bilateral and fairly equal.

The frontal sinus develops either as an "out-pouching" of an anterior ethmoid cell or as an extension of the infundibulum. It is slow in growth until the individual is about seven years of age, when it begins to increase in size. It reaches its full development at approximately twenty years of age.

The anterior wall of the sinus forms the prominent superciliary ridge above the eyebrow. It is usually quite thick and contains considerable marrow. In contrast to the anterior wall, the posterior wall is made up of thin, compact bone and separates the sinus from the anterior cranial fossa. There are numerous openings in the posterior wall, through which pass intercommunicating vessels that supply the membrane of the sinus and the dura. These openings serve as pathways for the extension of infection from the sinus to the meninges. The floor of the sinus is also composed of thin, compact bone and separates the frontal sinus from the orbit.

The frontal sinus communicates with the nose through the nasofrontal duct which opens into the middle meatus just under the anterior tip of the middle turbinate.

The sinus cavity is lined with mucous membrane containing ciliated columnar epithelium. The sinus is kept free of secretions by the action of the cilia, perhaps aided by gravity. Air pressures are equalized through the nasofrontal duct.

Acute Frontal Sinusitis—*Pathology*—Acute frontal sinusitis begins as a diffuse inflammation of the sinus mucosa with the usual phenomena of hyperemia, serous exudation, edema, and polymorphonuclear and lymphocytic infiltration. The channel of egress, the nasofrontal duct, may become closed or partially closed at this time due to swelling of its mucosa. Pus develops, forming an empyema of the sinus. Unless adequate drainage is provided the infection may extend from the mucosa to the bone. If this bone is compact, as in the posterior wall or floor of the sinus, an osteitis occurs, if the bone is diploic, as in the anterior wall, an

osteomyelitis develops. Extension through the posterior wall frequently results in intracranial complications, whereas extension through the floor results in orbital complications.

Etiology—The organisms usually encountered are the *Staphylococcus aureus haemolyticus*, *Streptococcus haemolyticus*, and pneumococci. However, any of the organisms harbored in the respiratory tract may be present. *Staphylococcus aureus haemolyticus* is the most frequent cause of osteomyelitis.

There are three principal exciting causes of acute frontal sinusitis: (1) upper respiratory infections, (2) swimming, and (3) trauma.

In partial obstruction, the symptoms are mild, whereas, in complete obstruction, the symptoms are extremely severe. In severe cases with complete or nearly complete closure of the nasofrontal duct, the onset of pain is sudden. The pain is located above the eye, is constant, is difficult to control with any medication, and is usually associated with irritability, anxiety, and generalized headache. In milder cases with partial closure of the duct, the pain is less severe, there is little or no edema, and considerably less tenderness. In this type of involvement the pain is usually present after the patient arises in the morning, persists with in-

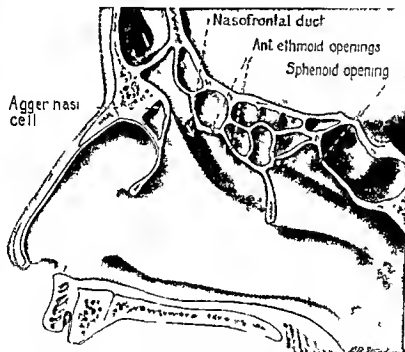


Fig 30—Nasofrontal duct and its relation

Any anatomic or pathologic process that interferes with the normal drainage of the nasofrontal duct is a *predisposing cause* of sinusitis. These include the following: (1) anatomical deformities (deviated septum and hypertrophied or misplaced middle turbinate), (2) allergy (allergic edema of the nasal mucosa may interfere with sinus drainage), (3) polyps of allergic or infectious origin, and (4) hyperplasia of the ethmoidal mucosa.

The frontal sinus may become involved through an ascending infection from the maxillary and anterior ethmoidal sinuses.

Symptoms—The symptoms vary in direct proportion to the amount of obstruction in the

creasing intensity until afternoon, and then decreases toward evening.

Examination—The findings also depend upon the degree of nasofrontal duct closure. If the closure is complete, such as usually occurs following swimming, the pain is extremely severe, the patient appears acutely ill, and the temperature may range upwards to 105° F. There may be considerable edema over the upper eyelid associated with injection of the bulbar conjunctiva. Exquisite tenderness is present especially along the floor of the sinus and over the supra orbital nerve.

In complete obstruction, intranasal examination may reveal little or no sign of disease unless

there is an associated upper respiratory infection or other sinus involvement Transillumination of the sinus usually reveals some opacity over the involved side Roentgenograms may or may not reveal cloudiness of the sinus If the film is exposed with the patient in the upright position a fluid level may at times be demonstrated The blood count usually shows marked polymorphonuclear leukocytosis

In partial obstruction of the duct, such as usually occurs in association with upper respiratory infections, the pain is less severe, the patient does not appear so acutely ill, and the temperature seldom exceeds 100° to 101° F Likewise, there is less tenderness and little or no edema over the involved side Intranasal examination reveals considerable edema, redness, and exudate in the middle meatus Transillumination and roentgen ray studies show practically the same condition as is noted in complete obstruction. The blood count usually shows a slight leukocytosis

Differential Diagnosis—The diagnosis of acute frontal sinusitis is usually not difficult It may be confused, however, with insect bite, lid abscess, acute ethmoiditis with abscess formation, supra orbital neuritis, or maxillary sinusitis

Insect bite is associated with marked edema but no noticeable tenderness over the sinus There are no intranasal or radiographic findings in these cases and there is usually a definite history of a bite

Lid abscess may be secondary to external trauma or associated with an acute ethmoiditis In the latter case the differential diagnosis may be difficult *Acute ethmoiditis with abscess formation* usually occurs in children and the pain and tenderness center over the medial orbital wall There is considerably more edema and intranasal exudate than in acute frontal sinusitis Transillumination and roentgenograms reveal a clear frontal sinus with haziness in the ethmoid area

In *supra orbital neuritis* the pain is usually irregular, sharp, and shooting in character It is distributed over the forehead and tenderness is confined to the distribution of the nerve There are no constitutional reactions or inflammatory changes in these cases

The pain in *acute maxillary sinusitis* may frequently be entirely frontal in character However, transillumination will usually reveal a dark antrum on the involved side, irrigation of which will alleviate the frontal pain

Complications—Complications are of necessity grave owing to the anatomical relationship of the sinus to the surrounding structures—notably, the meninges, brain, and orbit

A *subperiosteal abscess* may develop above or below the eyebrow because of extension of the pathologic process through the bone If the abscess forms above the eyebrow, it is indicative of an osteomyelitic process and offers a guarded prognosis If the abscess forms below the eyebrow (*orbital abscess*) it is indicative of necrosis of the floor of the sinus It may produce temporary acute proptosis This abscess is drained externally and the prognosis is good



Fig 31 —Acute frontal sinusitis.

Osteomyelitis develops in the frontal bone as the result of infection in the marrow spaces The prognosis is grave

The onset of *meningitis* is characterized by the usual signs of meningeal irritation These include nuchal rigidity, hyperactive deep reflexes, and characteristic spinal fluid changes The appearance of such meningeal signs indicates the sinus infection has extended through the posterior sinus wall to the dura, with involvement of the subarachnoid space

Persistent frontal pain or headache, after adequate sinus drainage has been instituted, is very suggestive of an *extradural* or a true *intra cerebral abscess* There is an increased spinal fluid cell count in these cases Brain abscesses secondary to frontal sinusitis are usually in the

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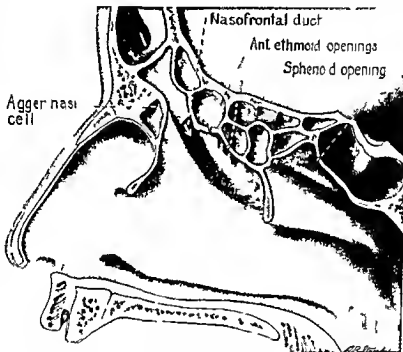


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frontal lobe but may occur anywhere in the brain

The most common complication of acute frontal sinusitis is the development of pathologic changes in the mucosa of the nasofrontal duct. This results in permanent interference with adequate ventilation and drainage of the sinus and lays the foundation for the development of a *chronic frontal sinusitis*.

Treatment—*Prophylactic measures* fall under two main heads

1 Caution in diving and swimming is imperative to prevent the sudden flow of water through the nasofrontal duct into the sinus

2 During upper respiratory infections, care should be used in blowing the nose so as not to force infection into the sinus. This is best ac-

commodated by a high blood concentration of the sulfonamide is essential. Penicillin may be more effective

3 *Rest* All patients with acute frontal sinusitis should be in bed until definite improvement occurs

Local measures include

1 *Physical measures* Heat over the involved sinus provides definite relief in the majority of patients. Some prefer cold in the first twenty-four hours

2 *Nasal shrinkage* Isotonic vasoconstrictors are indicated to reduce inflammatory edema and encourage ventilation and drainage of the sinus. These may be administered as drops or as a spray every two to four hours. It is desirable also to apply cotton pledgets of 4 per cent cocaine combined with 2 per cent ephedrine directly under the anterior tip of the middle turbinate to supplement the use of the nasal drops or spray

In the event of complete obstruction of the duct it is also advisable to apply 1:2000 adrenalin together with 10 per cent cocaine on cotton pledgets directly to the area of the frontal duct opening. This is done as an added measure in an attempt to relieve pressure within the frontal sinus

SURGICAL TREATMENT—If frontal pain and tenderness with or without edema persist and are *unrelieved by other measures* or if complications develop then the treatment should be surgical

There are three procedures which may be indicated in acute frontal sinusitis

1 *Elevation of the Middle Turbinate*—If there is persistence of pain unrelieved by other measures, simple elevation of the middle turbinate towards the septum is indicated. This may be sufficient to allow adequate drainage of the sinus as well as provide ready access to the nasofrontal duct opening for local applications and later irrigations by cannula

2 *Trephine Operation*—Before any operation is performed on the frontal sinus, roentgenograms should be made to determine the topography of the area

Under local or general anesthetic, an incision is made below the inner portion of the eyebrow at the inner angle of the frontal bone; this incision is made through the periosteum to the bone. The periosteum is elevated carefully over a very limited area so that no bone will be left uncovered after completion of the operation

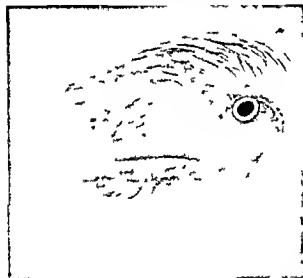


Fig. 32—Trephine opening in the floor of the frontal sinus—with tube in place

complished by blowing the nose with both nostrils open. Nasal shrinkage with an isotonic vasoconstrictor should be employed at regular intervals in an effort to maintain adequate nasal ventilation

General measures consist in the following

1 *Relief of pain* Patients with acute frontal sinusitis have considerable pain and adequate dosage of codeine or morphine is always desirable

2 *Chemotherapy* A sulfonamide or penicillin is indicated in all cases of total nasofrontal duct obstruction, as well as in cases of partial obstruction when associated with sudden onset and marked symptoms. In severe cases, since the organism is frequently a hemolytic staphylo-

An opening is then made through the floor of the frontal sinus, disturbing only the thin compact bone

If the sinus is entered through the superciliary ridge instead of the floor, marrow is encountered and secondary osteomyelitis may develop. The opening is made large enough to admit a fairly rigid rubber tube of not less than 8 mm in diameter. Usually pus under pressure will drain through the opening the moment the sinus is entered. The tube may be sutured to the skin to maintain it in position.

After the acute process subsides and purulent drainage ceases, the tube is withdrawn, provided the nasofrontal duct is open. The patency of this duct may be determined by forcing saline solution through the tube into the sinus. If the nasofrontal duct is open, the saline will pass freely into the nose.

3 Removal of the Posterior Plate of the Frontal Sinus.—The presence of meningeal signs in the absence of osteomyelitis is indicative of extension of infection through the posterior plate of the sinus. It is advisable in these cases to remove the posterior plate in order to obtain adequate drainage of the involved area and to remove the focus of infection. This is accomplished by enlarging the original trephine opening sufficiently to obtain adequate exposure of the posterior wall. The wall is then carefully removed, exposing the dura. A drainage tube is placed through the opening and held in position by a skin suture.

Chronic Frontal Sinusitis.—Etiology.—The exciting and predisposing causes of chronic frontal sinusitis are the same as those of acute frontal sinusitis. Chronic sinus disease develops as a sequela to one or more attacks of acute sinusitis.

Pathology.—The pathological changes may be limited to the mucous membrane itself or may involve both the mucous membrane and the surrounding bony structures.

The extent of involvement depends upon the virulence of the infection. The transition varies from hypertrophy and hyperplasia to actual destruction of mucous membrane. In the presence of continued suppuration, there is a change in a stratified epithelium with loss of cilia. Fibrinosis occurs in the submucosa with a resultant decrease in blood supply. This produces a non-functioning atrophic mucous membrane. These changes develop slowly and are accompanied

by a protective sclerosing process in the surrounding bone.

Symptoms.—The symptoms vary with the virulence of the organism, the local tissue resistance, and the degree of nasofrontal duct obstruction. Pain is usually absent, but occasionally, one hour or so after arising, a dull aching pain develops over the involved sinus area. This distress continues until afternoon when it usually disappears. The patient often complains of soreness and heaviness over the frontal sinus and is generally distressed and unable to concentrate. There is a continuous and often copious amount of purulent discharge which is usually more marked on arising in the morning.

Examination.—Examination usually reveals tenderness along the floor of the sinus and over the distribution of the supra orbital nerve.

The findings on intranasal examination vary with the severity of the frontal sinus infection and the amount of purulent discharge passing into the nose through the nasofrontal duct. Occasionally no abnormal condition within the nose is noted. The mucosa about the middle meatus is usually injected and covered by a thick, purulent exudate. Edema and thickening of the mucosa, with associated polypoid degeneration, are often present.

The frontal sinus is dark on transillumination due to the thickened mucosa. Likewise, roentgen ray studies reveal an increased density in the sinus as well as in the surrounding bone.

Differential Diagnosis.—The differential diagnosis is essentially that of headache in general. One must eliminate the possibility of ocular disease, supra orbital neuralgia, migraine, brain tumors and other intracranial lesion. Systemic conditions which might result in a toxic cephalgia also must be considered.

Intranasal findings, together with transillumination and roentgen ray studies, differentiate chronic frontal sinusitis from the above conditions.

Complications.—Complications usually develop following an acute exacerbation and are essentially the same as those of acute frontal sinusitis. Because the mucosa is already diseased, the increased pressure created within the sinus may result in an early extension of the infection to the surrounding areas.

MUCOCELE.—A mucocele is a retention cyst which develops because of obstruction in the ducts of mucous secreting glands or of the naso-

frontal duct This commonly occurs in the mucosa of a chronically infected ethmoid or frontal sinus. A history of previous surgery or trauma can frequently be elicited. The cyst increases gradually in size and may erode into the orbit by pressure necrosis. This produces a smooth swelling of the upper eyelid with downward and outward displacement of the eyeball. The cyst may become infected, thereby becoming a pyocele. Diagnosis is established by the above findings which occur in the absence of external inflammatory signs or symptoms. Treatment consists of surgical removal.

Treatment—The treatment of chronic frontal sinusitis is largely surgical. Such medical measures as nasal shrinkage, irrigations, and vaccines are mostly palliative and temporizing. However, recent investigation with the local use of penicillin and sulfa powders in the sinus has been very encouraging. Sulfonamides and penicillin should always be used prior to any surgical procedure for the correction of chronic sinus disease. This is done in an effort to prevent further extension of infection.

SURGICAL TREATMENT—Both intranasal and external surgical approaches are used in the treatment of chronic frontal sinusitis.

Intranasal operations are performed in an attempt to establish more adequate ventilation and drainage for the frontal sinus. Intranasal procedures include

1 **Correction of Anatomical Deformities**—These include (a) deviated septum by submucous resection and (b) enlarged or misplaced middle turbinate by partial turbinectomy.

2 **Removal of Polyps**—Polyps may be present owing to allergy or infection and should be removed.

3 **Anterior Ethmoidectomy**—The nasofrontal duct may open directly into the nose or may be tortuous and drain into the cells of the anterior ethmoid. The removal of these cells affords more adequate ventilation and drainage. The nasofrontal duct, however, should not be disturbed surgically because of the possibility of subsequent fibrotic closure.

4 **Maxillary and Ethmoid Sinus Surgery**—The maxillary, anterior ethmoid, and frontal sinuses open into the middle meatus. Infection in the maxillary and ethmoid sinuses results in inflammatory changes in the mucosa surrounding the nasofrontal duct. Therefore, treatment of these sinuses may result in better drainage of the frontal sinus.

External operations are performed to remove the infected membrane in its entirety and to establish a permanent, adequate opening between the frontal sinus and the nasal cavity.

There are four indications for this type of surgery.

1 **Relief of Continuous Pain**—If the more conservative forms of intranasal surgery fail to adequately relieve the patient's distress, extended surgery is necessary.

2 **External Frontal Fistula**—These fistulas form mostly during the period of acute exacerbation of a chronic sinusitis, with resulting blockage of the duct and rupture through the floor of the sinus. These can be closed successfully only by external frontal operations.

3 **Mucocele**—A mucocele must be removed in its entirety in order to prevent its recurrence.

4 **Complications**—External surgery is absolutely indicated in intracranial extension.

There are three principal external frontal operations.

1 **Lynch Operation**—At the present time this is the most widely used external operation on the frontal sinus.

Under local or general anesthetic an incision is made along the lower margin of the eyebrow and over the lateral wall of the nose. The periosteum is elevated from the floor of the frontal sinus and from the medial wall of the orbit. Care must be exercised in elevating the lacrimal sac, the inner canthal ligaments, and the attachment of the superior oblique pulley. As long as the relationship of these structures is not altered, there will be no change in their position postoperatively. The periosteum must not be buttonholed as it is elevated from the orbital bone because of the danger of introducing infection into the orbital tissue.

Bleeding from the anterior and posterior ethmoidal vessels can be readily controlled by electrocoagulation, pressure packing, or by ligature.

The entire bony floor of the sinus is removed, followed by complete removal of the mucous membrane lining the frontal sinus. Complete exenteration of the ethmoid cells is then performed, working from both the external and intranasal approaches.

The middle turbinate may be partially removed at the conclusion of the operation if it is enlarged or in any way obstructs the opening created between the frontal sinus and the nasal cavity. This opening may be maintained by

a mucous membrane graft, by the insertion of a large rubber tube drain or, as in many instances, left entirely alone

Sulfonamide powder is instilled into the sinus cavity, the periosteum is replaced and the skin closed by sutures

2 Killian Operation—This operation is indicated when there is considerable involvement of the anterior wall of the frontal sinus. The technic is similar to that of the Lynch operation

anterior wall of the frontal sinus. This results in obliteration of the frontal sinus and produces considerable external deformity

There are five factors which must be observed if success is to be obtained in external frontal sinus surgery

1 The entire bony floor of the sinus should be removed

2 The complete lining membrane of the sinus cavity should be eradicated

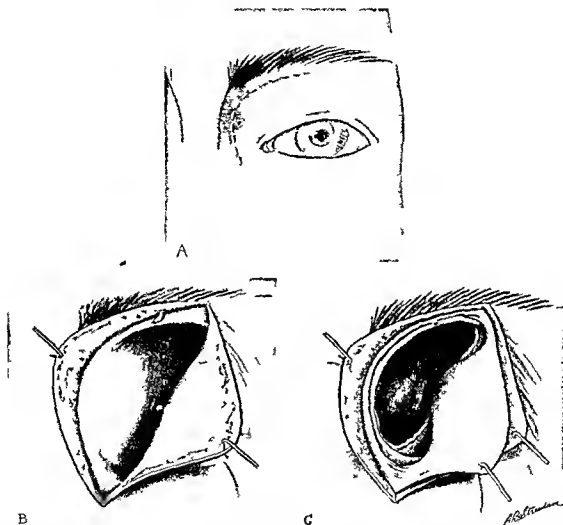


Fig 33—Steps in the Lynch operation A Line of incision on B Exposure floor of frontal sinus C, Floor of frontal sinus removed

but includes the removal of the anterior wall as well as the floor of the frontal sinus leaving the supra orbital ridge intact

3 Riedel Operation—This procedure is indicated when there is considerable involvement of the anterior wall including the supra orbital ridge. The technic employed is similar to that of the Killian operation except the supra orbital ridge is removed along with the floor and the

3 The ethmoid cells should be thoroughly extirpated

4 Pockets of mucosa or necrotic bone at the lateral extremity of the sinus should be completely removed

5 Coexistent disease in the contralateral frontal sinus must not be neglected

ETHMOID SINUSITIS

Anatomy of the Ethmoid Sinus—The ethmoid sinus does not essentially belong to the accessory sinuses of the nose as its entire labyrinth is confined within the nasal cavity (Fig 34). It is different from the other sinuses also in the fact that it consists of a number of cells or small cavities and its general appearance resembles the honeycomb. The cells are present at birth, and are formed by extension of the mucous membrane from the middle and superior meatus. The cells increase in size and number until about the twelfth or fourteenth year of life. They are paired, one occupying each side of the nasal cavity. Normally, they occupy almost the entire space between the septum and the medial bones of the orbit. The cribriform plate of the ethmoid lies between the capsule

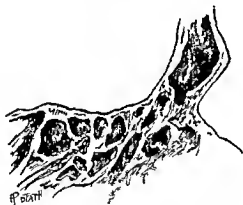


Fig. 34—The ethmoid capsule

and the septum. The capsule, which resembles a cube, is divided by bony partitions called lamella into two unequal divisions: the anterior portion and the posterior portion. These main divisions are likewise divided by bony partitions into other groups of cells. The number of cells varies from three or four to fifteen or more. All of these cavities are lined by mucoperiosteum of a pinkish color, covered by ciliated epithelium. Each cell communicates by its own ostium, either directly or indirectly, with the nasal cavity. Those of the anterior group drain anteriorly while those of the posterior group, through their various ostia, drain posteriorly into the nasopharynx. The arterial supply is from the anterior and posterior ethmoidal artery. The venous return is by the ethmoidal veins which communicate with the ophthalmic vein which drains into the cavernous sinus. The

nerves are from the nasal branch of the sphenopalatine.

Symptoms and Diagnosis of Ethmoid Sinusitis—The ethmoid labyrinth is involved to some extent in every case of acute coryza, rhinitis, or cold. Should this acute infection persist past its usual length of time for recovery, we then are likely to have some change in the membrane (intracellular or extracellular) which probably will be permanent.

The symptoms of acute ethmoiditis are those usually associated with acute coryza: a stuffy nose, inability to breathe properly through the nose, a stinging or picking sensation associated with the nasal membrane, headache of a dull character situated between the eyes and over the parietal region radiating toward the vertex. This headache is persistent and continuous. The movement of the eyes may be limited through associated swelling or toxic absorption. We may have edema or bulging of the skin between the inner canthus of the eye and the nose. Malaise, anorexia, fever, and general toxemia of a marked nature with increased leukocytes are always present. Inspection of the nasal cavity will reveal congestion of the mucous membrane of practically the whole area with increase of the congestion and swelling in the middle turbinate region. Pus or mucus will probably be seen over the inferior turbinate, this coming from under the middle turbinate. Radiographs are very valuable in showing the size, extent, and character of the labyrinthine anatomy.

Treatment—Treatment of acute and semi-acute ethmoiditis should be directed essentially to the establishment of adequate ventilation, drainage, and the restoration of ciliary activity.

Medical Treatment and Management—Medication should be applied by means of appropriate sprays, medicated packs, or tampons, or may be instilled by placing the patient in the proper posture for the displacement method advocated by Proetz.¹ The patient should be put to bed in a room of proper temperature and humidity. External heat (an electric pad or hot water bottle), a quartz light, or diathermy may be used for the relief of pain and congestion. Certain sprays for shrinking the congested membrane, of which cocaine (1 per cent or 2 per cent) in an isotonic solution is probably the best, can be decidedly helpful. Internal medication, consisting of a hydrogogue purge, some form of the salicylates, and tincture of aconite

in drop doses at stated intervals for the reduction of the temperature, is, in my opinion, far superior to the use of any of the coal tar products. Some form of mercury, usually red mercuric iodide in $\frac{1}{84}$ grain (0.001 gm.) doses three times a day, is also excellent. Since experience has shown that in all cases there is deficiency in calcium and iodine, these should be prescribed, and their use continued for some time. Unfortunately, treatment of sinus infections with the sulfa drugs has not come up to expectations. They must, however, be kept in mind.

Cultures should be taken of the nasal as well as the pharyngeal secretions, and treatment guided by the specific character of the infection present. A blood count, urine examination, and careful physical examination are of great value in obtaining a more complete knowledge of the patient, his normal resistance index and many other factors. The diet should be light but sufficient. Orange or other citrus fruit juice should be given in large quantities.

After the acute symptoms have subsided, and the patient has partially recovered, a complete roentgen-ray examination should be made. This will help to determine the extent to which the infection has radiated. One is now able to follow the advice of Skillern,² "Find the pus and trace it to its origin." Should the infection be limited to one or two cells, it is very easy, under local anesthesia, to infract the middle turbinate and use capillary suction directly in the diseased cells. Medication can then be introduced directly into the diseased area. This may be done either by the capillary spray or by the use of the tampon. Massive suction is not advocated since its use tends to create too great a vacuum which may favor changes in the mucous membrane. Capillary suction, on the other hand, is of undoubted value.

In a certain percentage of cases in which the discharge persists, even with good drainage and aeration, the Dowling pack with diathermy is effective. The cotton pack is wrapped on metal tape electrodes, saturated with a 20 per cent Silvol solution made in a 50 per cent aqueous-glycerine solution, or Sulfedex (Abbott), or Sulmefrin (Squibb). This tampon is tightly packed into the infected area. The negative electrode is placed on the patient's wrist or the back of his neck. The current is gradually advanced to the point of tolerance and kept at

this point for fifteen to twenty minutes. This should be repeated twice weekly.

Surgical Treatment—Surgical treatment must be considered (1) when the tissues have lost their normal appearance and use, and do not respond to local medication, (2) when inflammatory complications occur in the cranial cavity or in the orbit, (3) when a fistula exists, associated with either the frontal or the ethmoidal sinus, and (4) where there is a bone necrosis, cysts, growths, and foreign bodies within the labyrinth itself. Changes which take place in the tissue are divided into two classes, hyperplasia with polyposis and suppurative ethmoiditis. In the first division we have extensive changes in the mucous membrane and bone, causing partial or total occlusion of the nasal cavities. Should the polypoidal changes be few, these can be readily removed by the cold or hot snare. Should the hyperplastic or polypoidal changes be extensive, however, a complete exenteration of the ethmoidal capsule must be considered.

Many operations, both intra- and extranasal, have been devised and used for the purpose of completely removing all diseased tissue. The most popular and probably the best *intranasal operation* is the Mosher operation for exenteration of the ethmoidal capsule.³ This operation, performed under local anesthesia, is as follows. After the nose, upper lip, and vestibules of the nose have been made as aseptic as possible by iodine and alcohol, the membrane is thoroughly desensitized by proper application of a solution of cocaine and adrenalin, 10 per cent. Sufficient time must be given for these solutions to act. If large polyps are present these should be removed. A Mosher curet is then inserted into the nose and its point directed against the tissues directly in front of the anterior attachment of the middle turbinate (see Fig. 35A). Pressure is now exerted, the curet entering the anterior ethmoidal cells. These are broken down to the area of the uncinate process. At this point it is well to remove the anterior portion of the middle turbinate, thus giving a better view of the area and permitting better orientation. This removal can be done easily by punch forceps or scissors and a cold snare (see Fig. 35B). The curet is now reinserted with the beak or point backward, and with a sweeping downward and backward movement the various lamella and cells are broken through, the curet finally entering the sphenothmoidal fissure (see Fig.

35C) Frequently the sphenoidal sinus is opened by this technic. Removal of the rest of the overhanging turbinate is now performed by punch forceps or scissors and the remnants of the cells and diseased tissues are now removed and the area cleansed and made as smooth as possible (see Fig. 35D)

terior ethmoidal artery, vein, and nerve may be clearly seen and the artery and vein should be clamped and tied. The opening into the nasal cavity can be made with a chisel and punch forceps. There is now a clear view to facilitate the removal of the lamina papyracea and the ethmoidal cells. The middle turbinate is entirely

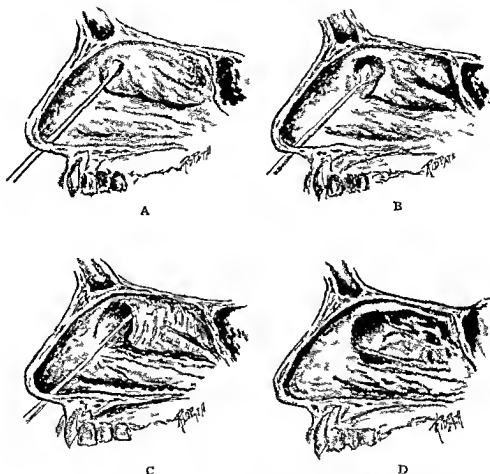


Fig. 35—Moshier's operation for exenteration of the ethmoid capsule

An *external ethmoid operation* may be performed should the intranasal operation be unsuccessful. Either local or avertin anesthesia may be used. The initial incision is made through the skin and underlying tissues in a slanting line from the eyebrow downward to terminate about $\frac{1}{2}$ inch below the inner canthus of the eye. The skin and fascia are elevated and the musculature divided. This exposes the small palpebral vessels which should be tied. These are then cut and the periosteum incised and elevated. The lacrimal sac is dislocated from its bed and turned inward. At this point the an-

terior ethmoidal artery, vein, and nerve may be removed and any spicules or roughness may be either wiped or cut away leaving a smooth area.

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SPHENOID SINUSITIS

Anatomy of the Sphenoid Sinus—The sphenoid sinus arises primarily from and is essentially closely associated with the cartilaginous nasal capsule. Genetically, it is demonstrable as early as the fourth month of fetal life. Shaeffer¹ states, "That it must be clearly understood that no portion of the fetal sphenoidal sinus is contained within the sphenoid bone—it is necessary that resorption of the intervening nasal capsule take place before the terminal nasal sinus or early sphenoidal sinus can come into actual contact with the body of the sphenoid bone." It is thus easily seen that the fetal sphenoidal sinus is a constricted portion of the nasal fossa and that the proximal end of the constriction remains as the ostium of the sinus and is therefore necessarily located in a superior position to any of the concha or turbinates which develop.

Up until the third year the sphenoidal sinus is nasal, not sphenoidal, in position, it is after this period that the rudimentary sinus exclusive of the ostium, is surrounded by bone, and finally enlarges into a sinus through reabsorption of the sphenoid body. This last stage takes place much later, toward the fourth or fifth year. The adult sphenoid is a cavity directly in the body of the bone and occupies almost a central position in the skull varying in size according to its development. The pneumatization is exceedingly variable, frequently extending into the pterygoid processes and the lesser wings of the bone. Should the pneumatization extend into the lesser wings and the anterior clinoid process, then the sinus encroaches upon the optic nerve. I have seen several cases in which the nerve was within the sinus cavity. If the pneumatization continues, very large sinuses with walls of paper thinness may result, should empyema occur in these cavities the orbital and cranial complications of such a development are readily understood. Although the frontal and maxillary sinuses are sometimes absent, seldom is there a complete nondevelopment of the sphenoidal sinus. The usual findings have been of asymmetrical cavities, one much greater than the other in extent. The average capacity seems well established by many investigators at from 5 to 6 cm.

The sphenoid sinus is comparable to a cube² having six aspects, namely, the anterior, posterior, superior, inferior sides, and the external

and internal walls. Of these, the most important rhinologically is the anterior, since it contains the ostium and is the only wall which may be used for operative measures. This wall is seldom perpendicular but points downward and backward in an angular direction, increasing in thickness as it descends. The ostium, situated in the upper third of the sinus, is visible only where there is a considerable space between the sphenoid and posterior ethmoidal cells. In other words, should the posterior wall of the ethmoid be the anterior wall of the sphenoid, then the ostium may be seen more or less in the median line, but should there be a sphenoidal ethmoidal fissure, then, according to its depth, the ostium will be situated in such a position that normal visualization of the opening is impossible.



Fig. 36—The sphenoid sinus

Closely associated with the superior wall are the right and left optic nerves and the optic chiasm, while above or slightly posterior, are the coronary sinuses and pituitary body in the sella turcica. Within the mucous membrane covering the antero-inferior wall we have the pharyngopalatine artery. This artery is quite large and when torn or cut causes frightful hemorrhage. The external wall helps to form a portion of the middle cerebral fossa and is thus in direct communication with the cavernous sinus and internal carotid artery. Unfortunately, as the sinus increases in size during development, this external wall becomes thinner and thinner, sometimes disappearing entirely leaving only the perosteum or a dehiscence in place while numerous perforations or openings are formed for the passage of small venous capillaries which anastomose with the cavernous sinus. Situated as it

is, any disease of the sphenoid sinus may and does give rise to a complexity of symptoms

Sphenoid sinusitis may be divided into two classes acute and chronic. The chronic infections are subdivided again into (a) those with free drainage, and (b) those with deficient drainage

Symptoms of Acute Sphenoid Sinusitis—As far as I can recall I have never been called upon to treat acute sphenoiditis *per se*, that is, without the rest of the nasal mucosa being involved. Many sphenoidal sinuses when opened have an intensely red appearance, the lining membrane being acutely inflamed, but with this condition there is always associated a general inflammation of the mucous membrane of the sphenoidal fissure and of the membrane covering the superior and posterior ends of the middle turbinates. It is doubtful, therefore, if there ever is an acute infection limited to the sphenoid sinus. The many cases that are acute exacerbation of a chronic sinusitis are the usual ones referred to the rhinologist.

The symptoms of acute infections associated with other choanal inflammation have a rather decided character. Subjective symptoms³ are pain, inability to concentrate, forgetfulness, aversion to mental or any other work, cough, lassitude or malaise, anorexia, lack of interest, and vertigo. Objective symptoms are discharge, coccosmia, crusts or pus on the pharyngeal wall with probably a proptosis, more or less, of the eye. The ocular symptoms may be an enlargement of the blind spot, scotoma, of the scintillating, spot, hair, or feather varieties.

While the majority of patients suffering from this disease have several of these symptoms, others suffer from peculiar symptoms seemingly unassociated with the sphenoid.

Pain is experienced as headache in various forms. It comes mainly from the pathologic condition present within the sinus and may be due to pressure, or to reflex or toxic manifestations. Pressure from any cause, whether the result of congested and swollen membrane impinging on neighboring membrane, or of a tenacious, mucilaginous pus filling the sinus cavity and occluding the ostium, is likely to produce headache symptoms which are closely similar. Other forms of headache are the so called vacuum headache or those where we have a negative instead of a positive pressure. It may be true that it is an impossibility to have a negative pressure or closed cavity without the

mucous membrane of the sinus being inflamed, but this does not prevent the mucous membrane of the sphenoidal fissure or a postethmoidal cell, through swollen tissue or polyp formation, from occluding the ostium of the sphenoid from outside. The headache is mainly situated in the occipital region, radiating fanwise upwards toward the vertex.

Reflex pain of toxic or other origin may extend over the mastoid region, simulating acute mastoiditis in character and severity.³ This pain, accompanied frequently with considerable muscular stiffness in the neck muscles, may extend downward toward the clavicular region or as far as the humerus scapular-clavicular articulation. This symptom, first mentioned by Sluder in his original paper on lower third headaches,⁴ has been further commented upon by Fenton and Fay.⁵ Later investigation has discovered the anatomy of the sympathetic nerve fibers associated with the carotid artery and sheath which is involved, pressure exerted over this region produces or increases sphenoidal pains when this sinus is diseased. Many other reflex pains have been found associated with sphenoidal disease sufficiently often to be added to the symptoms likely to be present. Two of these, which were originally brought out in a treatise of mine,⁶ are, first, a reflex pain felt over the canine tooth of the side affected, second, pain of a burning or continuous stab-like character felt at the junction of the soft palate with the anterior pillar of the faucial tonsil. A third, now added since it is encountered so frequently it no longer can be neglected, is a dull, sickening pain of continuous character felt over the pyramidal crest. Another pain which is felt a considerable distance away but cannot be classed as arthritic since it has occurred too frequently as an associated pain, is one of lancinating character over the instep. There may also be pain back of the eye which is dull in character, and feels as if pressure were exerted from behind forward. This symptom is not increased by pressure over the eyeball or lid.

Vertigo may be of either a continuous or in intermittent nature. It is manifested whether the patient is in a recumbent or an upright position, and may be so pronounced and distressing in character as to cause a fall, or, if occurring at night, the patient may feel that he must hold firmly to either side of the bed to avoid falling out. Should there be bilateral involvement and virulent toxemia, the vertigo may be character-

ized by a sensation of rotary as well as lateral motion. Rapid changing of the angle of the head or turning it produce such severe vertigo that the patient may have considerable difficulty to avoid falling. A peculiarity of gait is noticeable in some cases. Many patients complain of an optical illusion or instability when walking on the street, have a tendency to walk curbside, have difficulty keeping to a straight line, and the buildings seem to lean toward the center of the street.

Sleeplessness is another symptom. In spite of the general toxemia present, the majority of sufferers are unable to sleep with any degree of comfort. A feeling of something breaking or discharging into the throat periodically causes frequent awakenings with fears of suffocating and makes their nights miserable. This periodic discharge is typical of sphenoidal infection and is due to the fact that the swollen membrane of the ostium acts as a valve when the pressure within becomes sufficiently great to force an opening a certain amount of pus is discharged, the membrane then closes, to be forced open again when pressure is once more exerted.

Cough, a most persistent symptom and source of annoyance, is always present, it is spasmodic and of a throaty character. A sensation of tickling similar to that associated with hypertrophy of the lingual tonsil is felt. This sensation persists until a partial clearing of the mucus and pus is brought about by hacking and expectoration.

Malaise and forgetfulness with inability to concentrate or take interest in their surroundings, work or play are symptoms usually elicited.

The *objective symptoms* are few and consist mainly of a congested postethmoidal mucous membrane, swollen tissue in the sphenoidal fissure with possibly a discharge discernible in the olfactory fissure and on the pharyngeal wall, or, with or without the aid of a pharyngoscope, a discharge may be seen coming from the sphenoid ostium. A temperature of 99.5° or 101.5° F is usual for this infection.

Symptoms of Chronic Sphenoid Sinusitis — Perhaps no other sinus presents such a wide deviation in the subjective, objective and toxic symptoms as the chronically diseased sphenoid.

There are various *objective symptoms*. Should a secretion be present it is seen either coming directly from the ostium or clinging on the phar-

yngeal wall. It is a tenacious discharge of a dark yellow or greenish color which quickly changes into scale or scab formation and may, by capillary attraction, invade the olfactory fissure as well as the choana. The typical caecomic odor is perceptible on the breath and from the nasal chambers. Enlargement of the blind spot is also a condition to be looked for. Paralysis of the external ocular muscles may occur. The mucous membrane when cleaned will be found to be hyperplastic in nature.

The *subjective symptoms* will be divided into pressure symptoms, mental symptoms, ocular symptoms, toxic symptoms, olfactory disturbances, and general systemic disturbances.

Pressure pains radiating in various directions may be due to (1) pressure of the secretion or swollen membrane within the cavity, or (2) to mechanical pressure from the actual edema of the parts due to venous stasis, (3) to the presence in the cavity of benign or malignant growths such as polyps or angioma. Pain from the first two conditions flares up periodically, lasting from one to several hours, but never leaves entirely. Pain from growths in the sinus is a continuous one which is not relieved by position or ordinary medicinal treatment. Of these pains, headache radiating in the same manner as in acute sphenoid sinusitis (see p. 58) is present in the majority of cases. Pain back of the eye is more constant and pronounced. The character of these pains depends entirely upon the pathologic condition encountered, and may be described as dull, throbbing or semithrobbing, or both. It may be diffused, not corresponding to the area of distribution of any special nerve. Any physical exertion such as walking or running, or the use of tobacco or alcohol will increase this symptom.

Mental excitement seemingly adds oil to the fire. Cohabitation is practically impossible, due to the excessive mental excitement and the depressed general condition.

Mental and toxic symptoms should be considered together, since it is due to toxemia that there are disturbances in loss of memory, mental dulness, general depression, malaise, inability to concentrate, aversion to work and lack of initiative. Toxic symptoms usually occur at distal points. Of these, stiffness of the neck muscles, both at the sides and back, together with vertigo is probably the most frequent. Other toxic symptoms such as pain over the canine tooth,⁶ in the palate or glossopharyngeus mus-

cle, in the muscles of the leg, over the instep (simulating the pain of flatfoot), or *tinnitus aurium* with diminution of hearing, are less frequent, although any one or a combination of several may be the only diagnostic factors in a particular case.

Ocular symptoms are of various kinds. Frequently patients are referred to the rhinologist or sinusologist because of some condition of the eye or a disturbance of vision. Two cases, which I have reported,¹ were referred to us on account of sudden blindness in one eye, all other symptoms usually associated with this infection being absent. Examination, however, revealed an apparently acute exacerbation of a long standing chronic sphenoid infection. These two cases and four others of a similar nature, in which sudden blindness has been the only symptom elicited from the patient, have come under my observation in the past four years. Sudden unilateral blindness may be due to compression of the optic nerve in the optic foramen or to perineuritis or toxemia. Exophthalmos, with or without edema of the conjunctiva, may be present to a lesser or greater degree. In many of our cases the eyeball is completely dislodged from its socket. Hair, spot or scintillating scotoma and diplopia are frequent manifestations. In one of my cases, polyopia of seven images was the outstanding feature. Several cases have been referred to me which had been diagnosed as detachment of the retina. Proper opening of the sinus with elimination of the pus and toxic material resulted in the adhesion of the retina to its underlying tissue and the return of almost normal vision.

Olfactory disturbances are frequently the result of the mucous membrane in the olfactory fissure and that covering the septal side of the middle turbinate and septum being congested and covered with pus. The sense of smell is diminished or lost entirely.

The general symptoms are sore throat, laryngeal cough, aphonia, hoarseness, bronchitis, gastric disturbances such as loss of appetite and a tendency to vomit which may be due to gastro intestinal disorders or to backing and retching to dislodge the tenacious secretion and adherent crusts on the nasal pharynx.

Hepatic and gallbladder infection frequently occurs with a yellow-colored soft palate as a symptom. The condition is noticeable only when it is examined by daylight and not by our usual source of reflected light. The pain over

the instep found in acute cases of the disease is also associated with chronic cases.

Diagnosis—The etiologic factors in the history of an infection as given by the patient may be of considerable value in aiding one in the diagnosis of an acute infection, although of very little value or help in a chronic condition.

The diagnosis of sphenoidal disease cannot always be made by a normal routine examination. Even in the case of an apparently normal nose, the fact that a condition is present which may be caused by an infection of the sphenoid is quite sufficient, however, to direct attention to this area. Many times physicians are at fault when, without a most thorough history taking and examination, they give an opinion that there is no infection, no disease of this most important sinus.

To make the necessary examination requires time, patience and persistence, together with a scientific interest in arriving at a correct diagnosis. The first essential is to examine all parts of the nose, making mental or written notes of the conditions found. Next, the mucosa is shrunk with a weak cocaine solution, the shrinking must be complete, which requires fully ten minutes. A second examination is then made and the areas which have not responded to the first solution are touched up with a stronger cocaine solution (5 or 10 per cent). It will also be advantageous to apply this stronger solution between the middle turbinate and septum and anterior wall of the sphenoid (Adrenalin or other drugs of this character must not be used in making the examination). While waiting for cocaineization to be completed, a careful postrhinoscopic examination should be made, and the condition of the mouth, tonsillar areas, and larynx noted. It should be noted that in some cases of postnasal dripping the pus lodges in the pyriform sinus with resultant ulcerations, fistular formation, or perichondritis.

When cocaineization is complete, the pharyngoscope and a Skillern probe² (Fig. 37) are used to find the ostium of the sinus. As stated before the deeper the sphenoidal recess, the closer to the exterior the ostium will be located. It is frequently necessary to bend the probe at an angle to enter these sinuses. When the probe penetrates the sinus, a bead of white pus may be seen by the aid of the pharyngoscope placed in the other nostril and directed to the side being examined. Drainage continues along the shaft of the probe. In other cases, those of an anae-

robic nature, as soon as the probe enters the sinus cavity a slight noise may be heard by the patient and doctor which is caused by an inrush of air into the cavity, sometimes this is followed by an immediate cessation of all symptoms.

The position of the ostium having been located, the sinus can now be irrigated, the patient holding his head forward to prevent the lavage material from going down his throat. If a roentgen ray study is to be made, it can be done at this time with either the silver probe left in the sinus or a radio opaque oil injected into the cavity.

Complications and Sequelae—Thrombosis of the cavernous sinus may occur. Due to the intimate anatomical relation between the two structures, any infection of the sphenoid sinus may readily spread to the cavernous sinus. Retrobulbar neuritis followed by partial or complete loss of vision is another complication too frequently met. Basal meningitis develops more frequently than thrombosis of the cavernous sinus. No matter what the treatment, I have

necessary, the removal by surgical means of diseased mucosa or bone.

Medical Treatment and Management—A patient with acute sphenoid sinusitis should be confined to bed or to his room to prevent exposure to variations of temperature, and instructed in the proper posture he must keep to insure drainage. The intestinal tract is kept open by a hydrogogue purge. The diet should be light, and smoking or drinking alcoholic beverages prohibited. A prescription containing phenacetin, aspirin, strychnine sulfate and a slight amount of Dover's powder, administered at intervals, is excellent. Chemotherapy has not been of great value, but should be used if suited to the invading organisms. The space between the middle turbinate and septum should be kept open either by a spray of some solution to shrink the mucous membrane, or by infiltrated tampons properly placed. Calcium or calcidin should be given in proper dosage. An electric pad or other external heat placed over the orbit when the patient is in the proper position



Fig. 37—Flexible silver nasal probe, R. H. Skillern's graduated in centimeters on the shaft

never seen one of these patients get well. Occasionally a patient is treated surgically and a large opening made in the anterior wall with free drainage, yet after the complete disappearance of symptoms, in a short time some or all of them return.

Pharyngoscopic examination reveals that the opening which had been made is closed either by the mucous membrane acting as a curtain or by granulations which partially or entirely fill the sphenoid cavity, pus or discharge oozing from the sinus or tissue within. Where the mucous membrane has closed the opening of the sinus a secondary one must be made. In the case of granulations, the tissues covering the former opening are removed by cautery or electric desiccation.

Treatment—The aim of all treatment of sinus infections should be to bring the tissues within and surrounding the infected sinus back to normal as soon as possible. This includes the reduction of the inflamed or engorged mucosa, thus facilitating ventilation, drainage, and, if

—lying on his abdomen with the head low and the nose pointing toward the floor—may be helpful. Lavage of the sinus with warm normal saline solution may be tried. Autogenous vaccines may be used to bring resistance to a higher plane.

In my experience, however, I have never found any of the many measures enumerated to be a cure for this condition. This is, in all probability, due to the anatomic situation of the sinus and the position of its ostium. Patients improve but are easily reinfectd, or the membrane is so changed that a latent or lingering infection remains. It is with this knowledge and experience that I would advocate the removal of the anterior wall in all cases of infection involving the sphenoid sinus. A roentgenographic study should always be made before any operative measures are undertaken.

The Ridpath Technic for Removal of the Anterior Wall of the Sphenoid Sinus—This operation should be performed only under local anesthesia. It is advisable to prepare the patient

beforehand by administering 10 or 15 grains (0.65 or 1.0 gm.) of barbitol a half hour before the operation. The nose is sprayed thoroughly with a 2 per cent cocaine solution; this is repeated after five minutes. If after waiting an other five minutes the tissues are sufficiently shrunk and anesthetized to permit manipulation without pain, then cotton applicators saturated with a 10 per cent solution of cocaine hydrochloride to which a few drops of adren

of the septum corrected. If there is a good view and if by sounding the sinus the position of the ostium has been carefully ascertained, any one of the various punches may then be used to make the initial opening (Fig. 38-39). Should however the enlargement or hypertrophy of the posterior end of the middle turbinate interfere with the view or with aeration and drainage of the sinus, then this portion must be removed. Many and varied are the ways of doing this

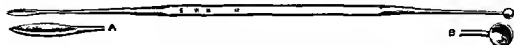


Fig. 38—Curet, Ridpath's. Double-ended, with round and pointed oval curets. Length over all 10 inches. Graduated in centimeters on the handle.

alin (1:1000) have been added are passed into the nasal cavity until they reach the sphenoidal area. These are allowed to remain in place or a second application made to the area until the membrane is thoroughly anesthetized.

A properly marked flexible Skillern probe (Fig. 37) the tip slightly bent is now passed backward bisecting the middle turbinate into the sphenoid recess. By gently rotating

but by repeated trials and experience the author finds the easiest and most practical way is by the use of the two pointed bevelled V shaped chisel of Hajek (Fig. 40). With the concave side of this chisel downward, one point placed under the inferior edge of the middle of the concha, the angle pointed so as to bisect the turbinate. Little pressure is needed to cut the tissue in the proper direction.

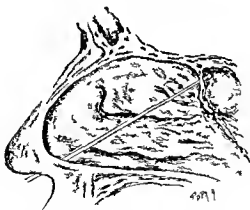


Fig. 39—Sounding the sphenoid sinus. The tip of the probe is bent slightly downward and outward and bisects the middle turbinate.

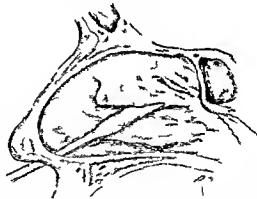


Fig. 40—Using the V shaped chisel for severing the middle turbinate where the posterior end obstructs the view.

and moving the probe the tip will, in the majority of cases, find the ostium and enter the sinus cavity. Should any difficulty be encountered and the probe fail to find the ostium, then the probe must be withdrawn and a greater curve of the tip made. When the probe enters the sinus it will be noted immediately, not only by the sensation of its entering a cavity, but also by the markings on the shaft of the instrument. Any obstructing masses, such as polyps or hypertrophies, are now removed and high deflections

There should be little or no pain or bleeding. If a pair of Luc forceps (Fig. 41) are now inserted and so placed as to grasp the cut end, a slight twisting motion is all that is necessary to detach the cut mucous membrane and remove it en masse. Very slight if any bleeding should result from this maneuver as the clamp of the Luc forceps and the twisting of the tissue en masse have torn the small arteries instead of cutting them.

There should now be a perfect view of the area to be operated upon. Figure 42 illustrates

the use of the author's sharp pointed curet employed in the initial puncture. This puncture should be made through the anterior wall as near the septum as possible. This position, if

not removed but all other parts of the anterior wall are. Unless there is some specific reason, such as a deep depression or cul de sac, the floor is left intact. In our work, all methods of op-

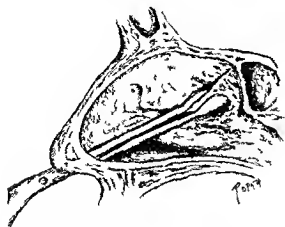


Fig 41—Removal of the detached portion of the middle turbinate by the Luc forceps

necessary can be visualized by consulting the roentgenograms. Placing the point on the selected spot, with a slight rotary motion with pressure, the anterior wall is easily penetrated and the sinus entered. Other features of this curet are the markings on the shaft, the thinness of the instrument which permits good view, and that, following the initial puncture, sufficient pressure may be exerted by depressing the distal end to break the wall downward thus facili-

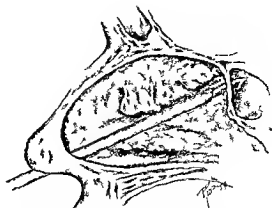


Fig 43—Illustrating the removal of the anterior wall. The Farachi bone-cutting forceps used

erating, saving mucous membrane flaps, suturing, tying blood vessels, have been tried, with the conclusion that other than in an external operation involving the frontal, ethmoid, and sphenoid, these are of little, if any, value

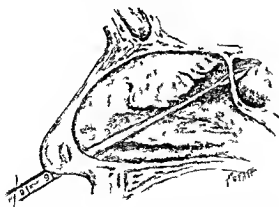


Fig 42—The initial puncture through the anterior wall of the sinus by the use of the author's sharp pointed curet

tating the removal of the remaining portion. The anterior wall is now removed by the Farachi (Fig 43) or other biting forceps, cutting toward the septum, laterally, superiorly and inferiorly. The ostium is not removed or traumatized. The tissues directly under the ostium are

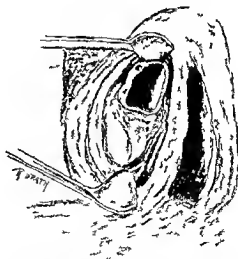


Fig 44—Showing the removed portion of the wall. The remaining portion with the ostium. The finished operation. The entire middle turbinate is omitted to improve the view

Figure 44 shows the completed operation, saving the ostium, the anterior bony wall, and the tissues lying directly under the ostium.

If polyps or other growths are seen within or protruding from the cavity, it is best to wait for twenty four to forty eight hours before re-

moving them. Unless there is very considerable hemorrhage do not pack, and if this should be necessary it is advisable to remove the packing within one or two hours. Owing to the likelihood of delirium (which is more frequent in this sinus than in any other part of the bony framework), curettage of the sinus mucous membrane is extremely dangerous. Even if the infected membrane is so edematous as to fill the entire cavity, it is surprising how quickly it returns to normal following the opening of the sinus.

After-treatment.—Unless some special condition manifests itself, or further operative measures are necessary, it is best to leave the area of operation alone for several days. Should the patient have fever, chills, postoperative bleeding, or severe occipital headache, the area should be looked over and if found at fault corrective measures instituted. In this respect the position of the patient in bed is of paramount value. The pillow should either be small or none at all used. The patient lies on his abdomen face down with his nose in the most dependent position. If necessary he should lie with his head over the edge of the bed. This factor is not considered as frequently as it should be and is probably one of the reasons for failure in the treatment of disease in this sinus.

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INTRACRANIAL COMPLICATIONS OF RHINOGENIC INFECTION

Meningitis.—Meningitis is probably the most frequent intracranial complication of rhinogenic disease. It occurs most often during the course of an acute lesion and is predominantly a disease of adult life. Its development may be insidious and the condition not suspected until it is already dangerously advanced. It has been stated that nearly 50 per cent of all cases of rhinogenic meningitis follow intranasal operations.

In the presence of acute infection, operations on the septum or removal of polyps should be avoided. Pneumococcal infection is by far the most common and occurs in about 50 per cent of the cases. Pneumococci of types III and IV are the most frequent offenders.

Rhinogenic meningitis represents a protective inflammatory reaction, which may result from infection in the accessory sinuses of the nose, particularly in the ethmoid or sphenoid. Inflammatory erosion of the roof of the ethmoid or sphenoid sinus is said to be a frequent lesion, but we have not been able to substantiate this concept except in cases of traumatic origin.

Pathways of Infection.—Infection of the meninges may result when micro-organisms enter the arterial stream from the accessory sinuses, the organisms being carried to all parts of the body and arrested in the small pial arteries. When infection around the veins of the mucosa occurs, bacterial toxins transmute through the vessel wall and, injuring the endothelial lining of the vessel, produce a thrombus, which becomes infected and advances by retrograde extension to cause meningitis.

There is no direct continuity between the lymphatics of the peripheral parts and the leptomeninges, but infection in the perivenous lymph spaces is carried to the lymph spaces around the larger veins and from these spaces the bacteria enter the lumen of the vein to reach the leptomeninges.

An intermediate pathway of infection may exist, as in meningitis which follows brain abscess, or there may be pre-*ex*ist thrombophlebitis of the cavernous sinus, the latter being, according to Turner and Reynolds, a rather frequent occurrence. Veins from the lining membrane of the sphenoid sinus enter the Haversian canal in the osseous wall of the cavity and pass directly into the cavernous sinus. The anterior

and posterior ethmoid veins enter the superior ophthalmic vein and so drain into the cavernous sinus

The meninges may become infected by invasion through one or several pathways simultaneously, in which event the meningitis is quickly generalized or is circumscribed for only a very short period. This is particularly true if the meningitis originates in the anterior or the middle fossa of the skull. This multiple invasion is most frequent in cases of rhinogenic meningitis in which the infection travels along the sheaths of the olfactory bundles.

Spread to the meninges may be a result of osteomyelitis and osteitis, lymphatic extension by way of perineural sheaths, lymphatic extension through perivascular lymph channels, vascular spread through perforating vessels to the cavernous sinus, direct invasion of the meninges through congenital dehiscence, or spread through a persistent cranio-pharyngeal pouch.

In only a small number of cases can a microscopic infectious lesion in the paranasal sinuses be demonstrated as a probable focus of infection. Microscopic bony defects, when they occur and produce an avenue for infection, are invariably traumatic in origin and most often result from intranasal operation.

That part of the ethmoid capsule which is adjacent to the dura is made up of lamellae of compact bone. There is no bone marrow in these olfactory laminae. Compact bone does not harbor infection and therefore meningitis from ethmoiditis of the anterior and middle group of cells is practically nonexistent.

Although dehiscences are frequent in the lamina papyracea and microscopic congenital dehiscences are not uncommon in the sphenoid bone, meningitis rarely occurs (except in cases of operative trauma) because the external layer of the dura is covered by mucous membrane, which is the protective mechanism of the nose and sinuses. Many postoperative infections travel through the lymphoid tissue of the nasopharynx, and infection in the nasopharynx may sometimes travel up through a persistent cranio-pharyngeal canal (Rathke's) to infect the body of the sphenoid bone and then the meninges.

The well organized exudate at the base of the brain and the thin, flocculent, purulent exudate that covers the pia in the sulci and on the surface of the brain represent the point of entrance of the infection and nature's effort to limit its

extension. This area lies over the ethmoid cells, the sphenoid sinus, and the basilar process of the sphenoid bone.

Symptoms—The symptoms of meningitis are influenced largely by systemic involvement, limited meningeal irritation, or increased intracranial pressure. The disease is frequently ushered in by a feeling of malaise. Headaches are common and are dull and persistent. There may be a dull, heavy sensation between the eyes in the cases of rhinogenic infection. Transitory severe neuralgias are common, and various palsies of the cranial nerves may appear. When a discharge from the nose has been present, it frequently diminishes, although occasionally it may increase in quantity. Vision may be impaired and examination of the eyegrounds shows evidence of intracranial pressure. The patient may or may not complain of pain in the neck but does not have stiffness of the neck until the cisterna magna becomes involved. As the disease progresses, the patient lies on his back with his eyes closed, in a state of semicomatose from which he is easily aroused, the elevation of temperature is moderate and the pulse relatively slow. The abdominal reflexes may be diminished, and there is a suggestive Kernig's sign. At this time there is only moderate leukocytosis. The spinal fluid is under slightly increased pressure at this stage and is usually cloudy, with an increased cell count, but may not show any bacteria. Chemical analysis of the blood and the spinal fluid may present a clue.

Diagnosis—Generally the diagnosis of meningitis is not difficult because the clinical picture develops fairly rapidly and is rather impressive. However, when pneumococci of type III are the invading organisms its development may be, and often is, insidious. An early diagnosis can be made by a thorough study of the spinal fluid and the blood.

SPINAL FLUID EXAMINATION—Of all the chemical and laboratory tests, the most important is the examination of the spinal fluid. Proper aseptic technique must prevail in the spinal tapping and the removed fluid must be received in a properly sterilized container. Determination of the chloride content and the finding of an increase in lactic acid, lowering of the protein content, and loss of reducing substances may be important adjuncts to diagnosis, but the presence or absence of an infecting organism, the increase or decrease in the cell count, and the spinal fluid pressure are the cardinal

factors used to determine the pathologic process and the patient's status

The type of meningitis present may be suspected from the appearance of the fluid. Cloudiness in itself does not mean generalized meningitis. Obviously it is of vital importance that the bacteriologic identification be established early, in order that specific therapy can be instituted. While the infecting organism is usually found later with ease, in the early stages the pneumococcus, streptococcus, meningococcus, and *Bacillus influenzae* are sometimes difficult to demonstrate, especially if the patient has already received some form of chemotherapy with bacteriostatic effect.

By the time acute meningitis is suspected clinically, the spinal fluid has already undergone characteristic changes. There is an increase in pressure. The fluid, at first slightly cloudy, rapidly becomes more turbid and purulent and contains increasing numbers of organisms and cells (polymorphonuclear leukocytes predominating) and an increasing quantity of protein and fibrin, either preformed or forming in the test tube. The sugar content is reduced early and rapidly falls. It must be emphasized that the test for sugar should be performed immediately after aspiration of the fluid because the sugar disappears *in vitro*. The chlorides are usually lowered. In serous meningitis there are no bacteria and the sugar content is normal.

DETERMINATION OF FOCUS OF INFECTION—The moment the diagnosis of meningitis is suspected, a thorough search must be made for an apparent focus of infection of rhinogenic origin. Frequently it is difficult to determine whether or not the sinuses are at fault and on which side of the head the apparent focus is situated. Even after a careful clinical and roentgenographic examination, one may be at a loss to know on which side to operate. It is in these cases of vague localization that a detailed history will be of great value.

DIFFERENTIAL DIAGNOSIS—Differentiation must be made from tuberculous meningitis, meningococcic meningitis, meningismus, serous meningitis, otitic hydrocephalus, cerebral poliomyelitis, encephalitis, subarachnoid hemorrhage, syphilitic meningitis, sympathetic meningitis, influenzal meningitis, torula meningitis, actinomycosis, suppurative epimeningitis, subdural hematoma, benign lymphocytic meningitis, primary sarcomatosis, secondary carcinomatosis of the meninges, and meningitis

produced by trauma, encephalographic examination, intraspinal introduction of serum, or induction of spinal anesthesia.

Treatment—**INDICATIONS AND PLAN FOR OPERATION**—If a search for a focal source fails to show at least some basis for suspecting the sinuses, then no operation should be performed, for the meningitis may be secondary to pneumonia or some other focal condition, in which case the additional physical drain of an unnecessary operation might contribute to a fatal outcome. But, there should be no delay in eliminating surgically a focal source, if there is any reason to suspect its existence.

In cases of rhinogenic meningitis, we are convinced as to the necessity of removing the diseased mucosa in the accessory sinuses, especially in the posterior ethmoid and the sphenoid and of the desirability of doing the operation by the external route.

SUPPLEMENTARY TREATMENT—Carefully controlled *cerebrospinal fluid drainage* is a widely used method of combating cerebral edema. The method should have application in treating the cerebral edema of meningitis. It is believed by some that this procedure also removes inflammatory products in helpful quantities and that re-establishment of a normal circulation of the blood and cerebrospinal fluid is promoted. There is the possibility that the periodic removal of cerebrospinal fluid facilitates the transfer of the sulfonamide drugs through the choroid plexus. The observed increase of these drugs in the cerebrospinal fluid after drainage and the subsequent clinical improvement of some of the patients suggest the value of maintaining a relatively normal level of intracranial pressure by repeated lumbar puncture.

We advocate the use of repeated small *transfusions of blood*, 150 to 200 cc for adults and 75 to 100 cc for children, the purpose being to stimulate the hematopoietic system and to help in reversing the abnormal spinal fluid status to one as near normal as possible. Repeated infusions aid in avoiding dehydration.

The primary action of the *sulfonamide drugs* is one of bacteriostasis; they reduce the rate of growth of bacterial cells and in some conditions kill a small number by interfering with their normal metabolism. Preceding the use of sulfanilamide and its derivatives, in 1937, bacterial meningitis proved to be almost invariably fatal. A successful cure in cases of this disease is now, reasonably, expected. All of the major

compounds of this group are effective when administered orally. Though there are differences in the rate of absorption, due largely to the differences in solubility of the compounds, the absorbed drugs diffuse to all tissues and body fluids. In any instance in which there is delay or doubt on the part of the laboratory in establishing or definitely confirming a bacteriologic diagnosis, it is generally advisable to begin treatment with sulfadiazine (2-sulfanilamidopyrimidine). A blood level of the amount of the drug administered usually should be over 20 mg. per 100 cc. of blood and may be stepped up to 40 mg. in those cases in which the infecting organism is the type III pneumococcus. Blood count, blood concentration of the drug, and urinalysis should be done at least every forty-eight hours. It is self-evident, however, that the intelligent use of sulfanilamide and its derivatives requires precise bacteriologic study.

Doses of the sulfonamide drugs vary with the severity of the infection and with the individual tolerance of the patient. In general, one gives 1 to 1½ grains (0.065 to 0.096 gm.) per pound (0.5 kg.) of body weight in twenty-four hours, the initial dose being about one-half the calculated twenty-four hour dose. If the patient cannot tolerate or is too ill to receive oral medication, the drug may be administered parenterally. Sulfanilamide in 1 per cent solution in sodium lactate may be given only subcutaneously or rectally, while the sodium salts of the other compounds may be given intravenously in 5 per cent solution in sterile physiologic solution of sodium chloride or in distilled water, as well as subcutaneously.

As to untoward reactions to chemotherapy, it is well to remember that individual tolerance to the drugs differs. If there is any previous history of drug sensitivity, a small oral test dose may be administered and about twelve hours allowed to elapse. In the absence of toxic manifestations, such as cutaneous eruption or of any increase in the symptoms of nausea, fever, and headache, which may heretofore have been present, intensive therapy may be instituted. In the order of their deleterious effects on the liver, sulfanilamide seems to be the worst offender, followed by sulfapyridine (2-*p*-aminobenzene sulfamidopyridine), sulfathiazole (para-aminobenzene sulfonamido thiazol) and sulfadiazine.

In some infections which do not respond to the sulfonamides, *penicillin* has occasionally produced spectacular improvement. The intra-

venous or intramuscular injection of penicillin (see "Cavernous Sinus Thrombosis") should be supplemented by the intrathecal administration of 10 cc. of an isotonic solution containing 1000 units per cc., since dependence cannot be placed on the penetration of penicillin from the blood streams into the subarachnoid space.

Specific serum, with the production of antibodies, is still, in our experience, a valuable adjunct in the treatment of meningitis and may be used in combination with chemotherapy. It is employed particularly in those patients who tolerate sulfanilamide and its derivatives poorly and those who show no favorable response to chemotherapy within forty-eight hours.

Cerebral Abscess—This condition may occur as a complication of diseases of the frontal, ethmoid, sphenoid, and maxillary paranasal sinuses, the frequency of occurrence being in the order named.

Any of the pyogenic organisms may produce a cerebral abscess but the most common is *Streptococcus haemolyticus*. Organisms in an abscess may be mixed or none may be found.

The lesion, when secondary to infections of the nasal accessory sinuses, usually involves the anterior pole of the homolateral frontal lobe, although it may be on the opposite side, extending backward, upward, or outward, external to and above the anterior horn of the lateral ventricle.

Symptoms and Diagnosis—In the early stages there may be only obscure symptoms or none at all, although usually a history of a mild chill or chilly sensations may be elicited. The pulse and temperature are at first elevated especially in children, although later there are often subnormal fluctuations of temperature of one or two degrees. Pain in the head is the most common symptom. It may be continuous or intermittent but it gets progressively worse, especially at night, and is unrelieved by any form of medication. There is nausea, lethargy, loss of appetite, rapid loss of weight, and constipation. The tongue is coated and there is a foul odor to the breath. High fever, stiff neck, and Kernig's signs are present only when meningitis accompanies the formation of the abscess. Vomiting may occur depending on the amount of pressure produced by the abscess. Periods of stupor, during which the patient can hardly be aroused, are a later manifestation.

Ophthalmologic examination may reveal dilatation of the homolateral pupil. Eyeground

changes, such as enlargement of the retinal veins, are present, and optic neuritis is noted when the abscess localizes in the frontal lobe.

Lumbar punctures frequently reveal a clear fluid under pressure. The cell count may be normal but is usually 20 to 100 or more per cu mm. Polymorphonuclear cells predominate before encapsulation takes place. When the abscess is well walled off the lymphocytes predominate. The blood count usually reveals a decrease in the red blood cells and hemoglobin, and there are always more than 15,000 leukocytes per cu mm of blood with a polymorphonuclear count of over 80 per cent. The blood pressure is increased and the pulse becomes slower owing to the progressive intracranial pressure. Localization and lateralization are usually difficult and the neurologic findings often are minimal, but diminished abdominal reflexes contralateral to the lesion and exaggerated knee jerks and ankle clonus are usually demonstrated. Focalizing signs are absent when the frontal lobe is involved unless the abscess becomes large enough or induces an edema of the surrounding structures. When the abscess or edema extends back far enough, aphasia develops when the "dominant" lobe is involved and there occurs paresis of the opposite extremities, first the upper then the lower, and last of all the face.

Unilateral loss of smell may be a helpful finding and roentgenograms of the sinuses valuable. When a previous operation has been performed, the operative site may suggest the side involved, although the abscess may develop in the frontal pole opposite to the operated site, or it may be bilateral. Exploratory puncture may be done when there are definite reasons for suspecting a frontal lobe abscess. A ventriculogram will show compression and displacement of the ventricle backward and downward.

An electro-encephalogram will indicate changes within the cranial vault as manifested by alteration of the normal electrical potential. It inconveniences the patient very little and may help to establish the diagnosis and localize the lesion.

SUMMARY OF PROMINENT CLINICAL SYMPTOMS

- 1 Headache especially at night, and usually located on the side of the abscess
- 2 Occasional vomiting
- 3 Chemosis and usually some eyeground change
- Optic neuritis when the abscess is localized
- 4 High temperature toward morning

5 Pulse rate usually in normal relation to temperature. In later stages of abscess, slowed pulse is definitely an indication of intracranial pressure.

6 Insomnia some mental dulness and poor memory, dependent upon the progress of the brain destruction.

7 Convulsions

8 Tactico-optical aphasia

9 Diminished abdominal reflexes, contralateral to lesion

10 Exaggerated knee jerk and ankle clonus usually demonstrated

Prognosis—The outlook is not good.

Treatment—All extracranial foci of infection must be eliminated. The sinuses, when involved, must be completely extirpated. When no improvement occurs within three or four weeks, which is about the length of time required for the process to encapsulate, the abscess should be exposed through a trephine opening and evacuated.

Cavernous-Sinus Thrombosis—Cavernous sinus thrombosis may be associated with acute or chronic sinusitis, but occurs most commonly during an acute exacerbation of a chronic sinusitis, or may be due to extension of thrombosis of the angular vein set up by a furuncle of the nose. It consists of a septic thrombosis and partial or complete obliteration of the cavernous sinus, its tributaries, and usually of other venous sinuses contributing to it. The infection begins in the nasal accessory sinuses and gets into the cavernous sinus by way of the facial or angular veins to the ophthalmic and thence to the cavernous sinus, also by way of the olfactory perineural sheath.

Pathology—There is an acute and chronic form of sinus thrombophlebitis. In the acute form there is a sudden obliteration of the cavernous sinus by the septic thrombus. The chronic form presents no venous engorgement because slow occlusion of the sinus has allowed collateral circulation to be established. The micro organisms responsible are usually streptococci and staphylococci producing an inflammation of the intima. The intima becomes swollen and roughened and in turn causes a narrowing of the lumen of the vessel, thus slowing the blood stream and inducing the formation of a blood clot. A clot in the cavernous sinus may extend outward to the orbit through ophthalmic veins or go to the opposite cavernous sinus through the circular vein.

Symptoms and Diagnosis—The patient usually appears desperately ill. Chills and fever

occur early in the disease and recur regularly. The temperature may be elevated up to 105° F. There is persistent pain in the orbit and side of the head in conjunction with progressive edema and chemosis of the upper and lower eyelids. Nausea and vomiting are frequent. Although the general debility is marked, the mentality seems to be unimpaired until the late stages of the disease.

Chemosis and exophthalmos are the classic signs of the disease. The eyeball is protruded first on one side, then on both. There is restricted motion of the eyeball and later paralysis of the eye muscles. There may be ptosis, strabismus, and pupillary changes because of involvement of the cranial nerves in the cavernous sinus. Dilatation of the retinal veins may be seen on ophthalmoscopy. The classic symptoms of exophthalmos and chemosis may not occur, however, if there has been time for readjustment of the collateral circulation. A high leukocytosis develops with an increased polymorphonuclear count. The blood culture is positive especially if the sample is taken before the institution of chemotherapy.

Diagnosis on the probability of invasion from an existing infection may have to be made in latent cases. It should be suspected when the above symptoms supervene during the presence of a paranasal sinusitis or nasal infection. Polyps or granulation tissue which obstructs drainage usually indicate the possible source of the infection.

Treatment—General supportive treatment is, of course, essential and should be supplemented by small blood transfusions. Hot fomentations may give some relief. If a definite focus can possibly be established, it should of course be eradicated, but this is very rare. Surgery, when it is attempted, usually consists in approaching the cavernous sinus through the orbital or temporal route with or without ligation of the internal carotid artery.

Until the advent of chemotherapy, these cases were almost hopeless but now increasingly frequent cures are being reported and we ourselves have seen two cases in which complete recovery took place. The sulfonamide compound should be given intensively as outlined in the section on meningitis. If the patient makes no definite improvement, penicillin should be tried.

Penicillin is a particularly effective bacteriostatic substance, especially against gram-

positive organisms, but exerts only slight effect on gram negative ones. Evidence suggests that it is superior to any of the known antibacterial substances in the treatment of *Staphylococcus aureus* infections. It is very effective in the treatment of infections due to the hemolytic streptococcus, pneumococcus, and meningococcus. It has been found effective in the treatment of infections due to organisms resistant or "fast" to the sulfonamides. Penicillin would seem to find its greatest field of application in the treatment of overwhelming infections due to those organisms which are susceptible to it. In the treatment of these conditions it may be administered intravenously or intramuscularly. When it is intermittently administered, larger amounts of the drug are needed than when it is administered by continuous intravenous infusion. Doses ranging from 10,000 to 20,000 units must be administered every one to two hours intravenously or intramuscularly in order to maintain effective and continuous bacteriostatic concentrations in the blood stream.

For continuous intravenous administration, 30,000 to 40,000 units of penicillin for each twenty four hours would seem to be an adequate dose. One-half the computed twenty four hour dose of the concentrated penicillin solution should be added to one liter of isotonic solution of sodium chloride or in a 5 per cent solution of dextrose, and 100 to 200 cc of the solution is administered intravenously at a fairly rapid rate, after which the rate of administration is regulated to between 30 and 40 drops per minute.

Administration of penicillin should be continued for several days after the blood cultures are negative, the infectious process shows evidence of definite regression, and the temperature has returned to normal. After several days, the dosage of the drug may be gradually decreased. When adequate doses of penicillin are used in the treatment of acute infections due to organisms which are susceptible to this agent, there is, according to current clinical reports, decided clinical and bacteriologic improvement within a period of forty eight to seventy two hours. Failure of improvement to occur within seventy-two hours suggests that the organism is not susceptible to this antibiotic agent.

In cavernous-sinus thrombosis the anticoagulants are of great value. Heparin is administered by constant intravenous drip and we have given 100 mg in 500 cc of saline, supplementing this

by giving dicoumarin by mouth in dosage of 300 mg in twenty four hours when the patient begins to show improvement. The heparin and dicoumarin are then discontinued. Chemotherapy should be maintained for a long time thereafter.

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INJURIES OF THE PARANASAL SINUSES IN WARFARE

Compound Fractures—In addition to the procedures to be described for the care of injuries to the paranasal sinuses, any associated wound of the soft tissues of the face should be dealt with at as early a stage as possible. As in other injuries of the face, excision of skin edges is unnecessary, but all dirt must be removed by scrubbing or by the use of a scraping spoon. After foreign bodies, loose fragments of bone, and debris have been cleared out, the wound is dusted with sulfanilamide powder. Whether it is closed or left open will depend upon its position, its extent, and the degree of infection; secondary repair may be required.

Immediately after the accident, no extensive operation or opening up of fresh bony layers

should be attempted, such an operation as removal of the walls of the ethmoid cells or drainage of the maxillary or frontal sinuses, should be postponed until two weeks have passed.

Contusions—Blows over the walls of the sinuses or falls resulting in injury to these regions may cause hemorrhage into the sinus cavities. The only subjective evidence of hemorrhage from the nasal sinuses is escape of blood from the nose, but even this symptom is not invariably present. Transillumination and radiography will aid in the diagnosis.

Treatment should consist in restraint from any direct interference. Lavage is not required unless there is evidence of infection, which is, however, unlikely to occur spontaneously and is more liable to be caused by surgical interference. It is wise to apply an antiseptic, preferably alcohol, to the vestibules of the nose, and to require the patient to wear a pad of sterile gauze over the nostrils, held in place by tapes tied around the head. Steam inhalations may be useful to clear the nose, but syringing or douching must be forbidden. The blood in the sinuses will, in all probability, be removed by eiliary action with no harmful sequelae.

Fractured Base—A fracture through the floor of the anterior cranial fossa may pass through the roof of the ethmoid cells or across the cribriform plate. The accident is serious, especially if the dura is torn, because a route is afforded by which infection may reach the meninges.

In addition to any other evidences of fracture, there will be bleeding from the nose and possibly an escape of cerebrospinal fluid. The latter is distinguished from ordinary nasal secretion by the absence of mucus and albumen and by its ability to reduce Fehling's solution.

No direct treatment is required except careful antiseptics of the nostrils as described under "Contusions" and the giving of prophylactic doses of a sulfonamide for a few days. Blowing the nose, syringing, douching or packing must all be avoided. The cleft will probably close spontaneously, but if it remains patent, meningitis is almost certain to occur when the nose becomes infected. If after two weeks there is continued cerebrospinal rhinorrhoea, an operation should be performed consisting of elevation of the frontal lobe by external approach with the application of a graft of muscle or fascia lata to the upper surface of the cleft.

Fracture of the Walls of the Sinuses—When the face receives a direct blow from an antero-posterior direction the nose usually takes the brunt of the assault. The weak nasal bones readily yield, and when the force is sufficiently violent the strong strut formed by the nasal process of the maxilla gives way and is forced backward against the *ethmoid cells* crumpling the lamina papyracea. The frontal process of the maxilla on each side may break causing depression of the face.

Sometimes a strain is thrown on the strong rim of bone forming the lower margin of the orbit, this breaks at its weakest spot in the region of the infra-orbital foramen. A fracture then runs down the thin anterior wall of the maxillary sinus. The strong alveolus may escape but is, in some severe cases, pushed backwards with the rest of the central facial framework.

In spite of considerable edema early operation is desirable for elevation of the depressed bones which can usually be accomplished with the aid of Ash's and Walshman's forceps but in some cases the alveolus must be grasped with strong lion forceps or with those designed by McIndoe.¹ To keep the bones in position fixation to the mandible by approximation of the teeth with cap splints is sufficient. Sometimes however, a plate fixed to the head with plaster, carrying a bar for attachment to a denture, is necessary.¹

Fracture of the cribriform plate or the roof of the ethmoid cells may have occurred in association with this type of injury. In such case no manipulation to the maxillary bones must be attempted lest meningitis be precipitated.

As a result of a blow directed laterally the malar bone and often the outer wall of the maxillary sinus may be fractured; the eyeball may be displaced or the skull fractured. Replacement is effected by inserting a lever under the zygoma through an approach in the temporal fossa. No fixation is required.

If the anterior wall of the antrum is depressed and cannot be replaced by utilizing the methods described, a sublabial incision may be required to permit the insertion of a lever or chisel in the fracture line or through a free opening into the antrum. Following this procedure the sinus is sometimes packed for one or two weeks. Fractures not replaced before the end of two weeks may require continuous traction or correction by grafts. Fracture through the anterior wall of

the frontal sinus is not of great moment unless there is severe depression. For both cosmetic and functional reasons it may then be desirable to insert a lever through a small external incision for elevation of the bone. When the posterior wall is fractured the resulting communication between the nose and the interior of the cranium is a potential source of danger, particularly if the dura is torn. Antiseptic care of



Fig. 45.—Fragment of H. E. shell in pterygomaxillary fossa. The subject was a soldier injured in the retreat to Dunkirk. A missile had penetrated the ethmoid cells and had passed through the posterior wall of the maxillary sinus to lodge in the pterygomaxillary fossa. It was removed after an interval and with some difficulty by the transmaxillary route: there was considerable hemorrhage from the sphenopalatine artery during the operation. The foreign body produced infection of the maxillary and frontal sinuses and drainage operations were necessary to restore health.

the nostrils is required and the other precautions referred to under 'Contusions' must be taken. One of the sulfonamides should be given by mouth. Roentgenograms will show air in the anterior fossa. Cerebrospinal fluid may escape. If the cleft does not quickly close, it must be repaired by applying a muscle or fascial graft. Approach may be made through an osteoplastic flap lateral to the sinus or directly through the anterior wall. In the latter case it is advis-

able to obliterate the sinus, closing its duct with a small muscle graft

Penetrating Wounds—The entry of bullets or fragments of shells into the sinuses is likely to be associated with penetration of the cranium and is, therefore, fatal in many cases. Of those patients who reach the hospital the injury is caused in most instances by a missile traveling from the lateral direction. It is the maxillary sinus which is most likely to be wounded, but the ethmoid and occasionally the frontal sinus



Fig. 46—Fragment of trench mortar shell in maxillary sinus. The soldier was in a tank when hit. The shell fragment lay in the alveolar groove of the maxillary sinus. It was removed through an opening in the canine fossa fourteen days after entry. The antrum was not grossly infected but it was thought advisable to make a drainage opening through the lateral nasal wall by the Caldwell-Luc method. There was no subsequent disability.

may suffer. In addition to the damage to the sinus there may be injury to the orbit or eye ball, to the alveolus or mandible, and to the nasal septum or turbinal bodies.

The ordinary treatment for soft tissue wounds is required, together with removal of the causative object. A sublabial operation may be essential as a means of approach, there is no great hurry for its performance.

A piece of metal lodged in the depths of the ethmoid labyrinth in the cavity of the sphenoid sinus, or in the pterygomaxillary fossa is diffi-

cult to approach, the operation for its removal is best delayed for at least two weeks from the time of injury.

In the case of a foreign body in the ethmoid or sphenoid sinus the usual external ethmoid approach is best, the pterygomaxillary fossa is reached by a transantral operation.

In addition to the local measures referred to patients with severe wounds of the sinuses require treatment for associated shock, for any rise of intracranial pressure, and for wounds elsewhere. Hemorrhage may require control by pressure applied by means of iodoform ribbon gauze packing or possibly by ligation of the external carotid artery.

Complications—*Osteomyelitis* is less likely to arise spontaneously than as a result of precipitate and extensive operation. The local use of sulfanilamide powder, or of iodoform if preferred, helps to prevent its occurrence. The same remarks apply to *thrombophlebitis*, which usually results from operations performed during the early stages of local sepsis, thrombosis of the cavernous sinus and meningitis are possible sequelae. *Meningitis* is guarded against by conservative treatment and by the local and general use of sulfonamides, when symptoms appear, penicillin or intensive chemotherapy is instituted.

Extradural abscess may follow compound fractures of the walls of the frontal sinus and necessitates a radical operation. *Cerebral abscess* luckily is rare, as its occurrence in the anterior cranial fossa denotes a poor prognosis.

V E NEGUS

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TUMORS OF THE NOSE AND ACCESSORY SINUSES

It would be impracticable to enumerate all of the diagnostic agents, radium technics, and surgical procedures which have had a share in the advancement of knowledge concerning the treatment of neoplasms of the nasal cavities and accessory sinuses. In recent years, probably no factors have contributed more to the effec-

tiveness of treatment than have the increasing appreciation among medical men of the need for a histologic diagnosis of all tumors in this region, microscopic examination of fresh frozen sections of tissue at the time of operation, and the employment of surgical diathermy and radium in the eradication of these growths.

Many nasal tumors possess certain physical and roentgenographic attributes which, in a measure, are diagnostic. These characteristics, however, are not unfailing signs by which recognition of the nature of the growth can be assured, on the contrary, reliance must be placed on the microscopic findings if the final diagnosis is to be free from uncertainty. Too often biopsy demonstrates that the most innocent appearing mass in the nose or sinuses is in reality a highly malignant neoplasm. The value of diagnoses made from fresh frozen sections of tissue at the time of operation cannot be overestimated. Treatment of many diffuse and particularly low grade malignant neoplasms of the nose or sinuses is likely to be inadequate and unsatisfactory unless specimens can be removed for biopsy and immediate diagnosis at frequent intervals during the operative procedure to determine when the growth has been entirely eradicated. It is evident, therefore, that close cooperation between the surgeon and a competent pathologist is essential if creditable results in the treatment of tumors of the nose and sinuses are to be secured. Surgical diathermy has changed the management of some growths in this region from crude attempts at removal to dependable techniques of treatment. Not only does this form of electrocoagulation reduce the possibility of recurrences but also is of value in controlling the hemorrhage attending the surgical extirpation of many of these tumors.

Benign Tumors—A true benign neoplasm is a new growth composed of cells which resemble normal tissue cells but which fulfill no physiologic function. Inflammatory swellings and hyperplastic growths, on the other hand, originate as physiologic responses to some form of irritation. Since neither by clinical nor by laboratory methods is it always possible to distinguish a true benign neoplasm from an inflammatory or hyperplastic overgrowth, any abnormal mass of tissue in the nose or sinuses must be interpreted as a benign tumor if it lacks the infiltrative qualities and the power to metastasize characteristic of a malignant lesion.

Benign tumors of the nasal cavities and sinuses may be entirely asymptomatic or may produce varying degrees of nasal obstruction. Some may cause hemorrhages and a few, external deformities. Objectively, they vary greatly in size from tiny, inconspicuous lesions to masses which fill an entire nasal cavity. Owing to pressure necrosis of the adjacent bony walls, those of large dimensions which originate in the nose may invade one or more of the sinuses, similarly, those arising in a sinus may encroach on the nasal cavity.

Benign tumors may be pedunculated, sessile, or polypoid with a smooth, granular or ulcerated surface. They may be vascular, avascular, soft, friable, firm, or hard. Those of a dense character and more particularly those possessing cartilaginous or bony tissue are visible on roentgen examination. Each benign tumor produces certain combinations of the clinical findings just enumerated. Although the association of various signs and symptoms may be suggestive, it does not offer conclusive evidence of the nature of the lesion. The final diagnosis must be based on the microscopic findings.

The treatment of benign tumors of the nose and sinuses is based on the size, location, and nature of the growth. We shall consider the treatment of benign tumors in the nasal cavities and in the antrum in general and then we shall take up the various benign tumors which affect the nasal cavities and accessory sinuses. The characteristics which are peculiar to each type of tumor and special methods of treatment which deserve emphasis will be mentioned.

Treatment of Benign Tumors of the Nasal Cavities—Small, accessible tumors may be removed with biting forceps after which the base of the lesion should be touched with surgical diathermy. Local injection of procaine hydrochloride and topical use of cocaine or intravenous injection of pentothal sodium offer satisfactory anesthesia. Microscopic examination of all tissue removed should not be neglected. Larger and inaccessible tumors are best removed with the patient under gas and ether anesthesia. The anesthetic agents should be administered through an intratracheal tube, which maintains an airway in spite of bleeding into the pharynx. If the growth is not particularly vascular, biopsy may be performed prior to the operative procedure. Extremely vascular tumors, however, should not be disturbed until the tube has been inserted into the trachea for

the anesthesia. Histologic diagnosis of the lesion then may be made by the fresh frozen section method. Although avascular tumors may be removed with biting forceps, curets or snares, it is advisable to touch the raw surface from which the growth originated with surgical diathermy in an effort to prevent recurrences. Administration of ether during application of surgical diathermy should be stopped. Vascular

the surrounding normal tissues. This method of treatment maintains a comparatively dry field as the growth gradually is destroyed.

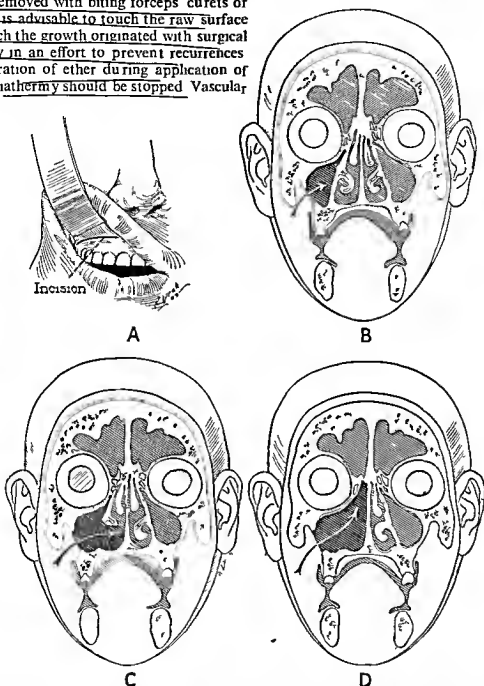


Fig 47—Transantral route for removal of the tumors of the antrum, nose and ethmoid region. A incision. B approach to antrum—if the tumor is of low grade it may be completely removed with surgical diathermy. If it is of high grade, radium may be inserted through this opening. The opening should be at least 2 by 3 cm. in size to allow for free drainage. C approach to tumors involving the turbinates and lateral wall of the nose which can be completely removed with surgical diathermy. D approach to tumors of the antrum, nose and ethmoid regions illustrated in Figure 48.

tumors are best destroyed by electrocoagulation using a small amount of current which is carefully controlled in order to prevent injury to

The removal of some bony tumors requires a chisel and mallet.

Whereas the majority of benign tumors arise

log in the nasal cavities can be removed by an intranasal approach, some require special surgical exposures which provide greater accessibility and better visualization of the growth

through an intra oral incision in the upper bucco alveolar sulcus and in which a portion of the mesial wall of the antrum is removed by means of bone forceps, facilitates removal of

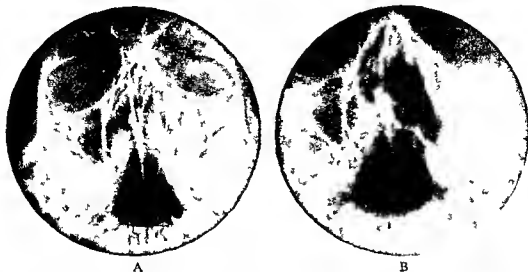


Fig 48—Papillary squamous-cell epithelioma, grade 1 of the right antrum, nose and ethmoid region, A, preoperative view, B, postoperative view—the tumor has been removed by transantral route by means of surgical diathermy No recurrence two years later

These methods of exposure are as follows (1) Lateral rhinotomy, in which unilateral, external incision is made through the entire thickness of the base of the nose, permits upward

some diffuse, benign tumors situated along the floor of the nose or in the ethmoid region^{1, 2}

Treatment of Benign Tumors of the Antrum.—Benign tumors of the antrum require a Cald-

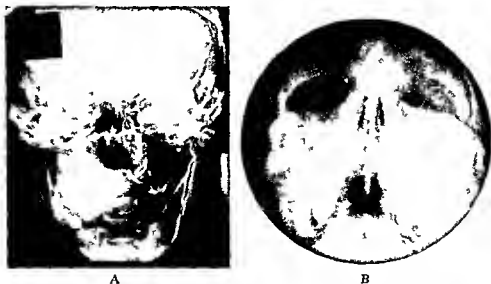


Fig 49—Osteoma, A, of the right antrum and upper jaw, B, of the right upper jaw and antrum and malar bone of a child seven years old The tumor shown in B was trimmed down through an incision underneath the upper lip so that both sides of the face were made symmetrical

retraction of the external portion of the nose for greater exposure of the affected nasal cavity (2) The transantral approach (Figs 47, 48), in which the interior of the antrum is exposed

well-Luc type of operation so which the interior of the antrum is exposed through a horizontal incision in the upper bucco-alveolar sulcus, a portion of the bony wall of the antrum in this

region being removed with a chisel. By such exposure the tumor can be removed with forceps, curets, or surgical diathermy if necessary.

Papillomas—Two types of papillomas arise in the nose: (1) hard papillomas which are warty lesions that occur around the nasal vestibule and can be destroyed with diathermy and (2) soft or mucoid papillomas which are inflammatory cauliflower-like structures which bleed and produce obstruction. The second type of papilloma is a fibrous tumor covered with hyperplastic epithelium; it contains many plasma cells and may involve the nasal cavities and sinuses. Surgical diathermy offers the best method of eradication.

Polyps—Nasal polyps, which are inflammatory, fibrovascular growths, are probably the



Fig. 50—Osteofibroma of the right antrum with calcification throughout the tumor and destruction of the outer wall of the right nostril.

most common tumors of the nasal cavities. The majority of them arise in the ethmoid region. Grossly, they are pedunculated, occasionally ulcerated, and sometimes hemorrhagic. Microscopically, they can simulate papillomas, fibromas, hemangiomas, and plasmocytomas. Occasionally, growths which appear clinically to be typical bleeding polyps are found to be pyogenic granulomas.

Adenomas, Lipomas, Fibromas, and Myxomas—Adenomas, lipomas, fibromas, and myxomas are extremely rare nasal tumors which can grow to fill an entire nasal cavity. On the septum, fibromas are likely to involve the anterior third, whereas myxomas occur more commonly in the middle and posterior thirds. The treatment of these four types of tumors should consist in destruction with surgical diathermy.

Lymphangiomas—Lymphangiomas are uncommon nasal tumors. When they do occur, they are best destroyed by electrocoagulation.

Hemangiomas—Hemangiomas are difficult to distinguish clinically from bleeding polyps and from granulomatous polyps. These growths are either capillary or cavernous. The capillary type is a sessile or pedunculated, dark red growth which occurs most frequently on the septum. It should be destroyed with surgical diathermy. A careful microscopic examination is desirable to distinguish capillary hemangiomas from hemangio-endotheliomas, for a vascular tumor of this type occurring after the patient is forty years of age is likely to be malignant.

Cavernous hemangiomas arising in the lateral wall of the nose are serious tumors. They grow by infiltration, destroy the surrounding tissues, and cause profuse epistaxis. They can fill the nose and sinuses, erode the bone to enter the orbit, and cause exophthalmos. Irradiation is the therapeutic method of choice until the tumor is scarred sufficiently to permit surgical removal.

Basal Fibromas—Intranasal hemangiomas must be distinguished from intranasal extensions of a juvenile basal fibroma. Basal fibromas which arise in the nasopharynx are true vascular fibromas. They usually arise in the vault of the nasopharynx from the periosteum covering the basilar process of the occipital bone and the body of the sphenoid bone. They also arise occasionally from the anterior aspect of the upper two cervical vertebrae, the internal pterygoid plate, and the pterygoid maxillary fossa. These hard tumors which are covered with normal mucous membrane can grow to fill the nasopharynx and extend into one or both nasal fossae. By pressure necrosis of bone, they can invade one or more of the sinuses, particularly the antrums. These fibromas appear about the age of puberty and tend to undergo spontaneous regression when the patient reaches the age of twenty to twenty-five years. Surgical removal of these tumors should be avoided because of the profuse hemorrhage attending such operations. Implantation of radium or electrocoagulation combined with radium therapy offers the most effective form of treatment. In many instances, repeated treatments of this type are necessary to eradicate the growth. When the antrum becomes invaded, a transantral approach to the part of the tumor in the antrum

and nasal cavity is recommended. By this exposure this portion of the growth can be removed readily with surgical diathermy.

Chondromas and Osteochondromas—Chondromas and osteochondromas are rare nasal neoplasms. Chondromas occur most commonly in the septum while osteochondromas are likely to involve the sinuses. Clinically the appearance and symptoms of chondromas are indistinguishable from those of osteomas. Chondromas however have a great tendency to recur unless they are destroyed completely. Thorough removal is more certain when surgical diathermy is employed.

Osteomas (Figs 49-50)—Osteomas are of three types: eburnated, spongy and mixed. Those of the eburnated type are composed of spicules of adult bone. Spongy osteomas are composed of ossifying fibrous tissue. All three types occur relatively commonly in the bones of the face and in the frontal bones. In order of frequency, osteomas involve the frontal sinus, ethmoid sinus, antrum, and then the sphenoid sinus. Some osteomas are true neoplasms and others are on an inflammatory basis. They can produce nasal obstruction, occlusion of the eustachian tube or exophthalmos. The cribriform plate of the ethmoid bone may become eroded and meningitis or brain abscess may ensue. Because of the number of instances in which osteomas combine the clinical features and etiologic characteristics of giant cell bone tumors and osteitis fibrosa, it has been concluded that these two conditions may be closely allied and that the possibility of a common cause must be considered. Some sort of trauma seems to be the most important etiologic factor.

It is important to distinguish osteomas from exostosis for exostosis does not tend to progress and may be treated by simple removal with chisels of the thickened portions which produce the deformity. Osteomas are neoplasms of unlimited growth and must be removed to gether with their base by surgical diathermy.

Cysts—Mucous cysts occurring in the floor of the nose and in the antrums require no discussion here. Simple surgical incision is all that is required for their removal.

Intranasal Gliomas—Gliomas are exceedingly rare tumors in the nasal cavity and usually originate from the cribriform plate of the ethmoid bone.

Malignant Tumors—The successful treatment of malignant neoplasms of the nose and

sinuses as in any other region of the body is based on early diagnosis and proper management. Unfortunately carcinomas of the nose and sinuses may become large before they produce symptoms of discomfort or before they are recognized clinically.³ Unfavorable results in the care of these lesions are based on one or more of the following factors: (1) poor visualization and inaccessibility of the growth; (2) failure of the clinician to recognize a malignant tumor until it is extensive; and (3) insufficient or unsuitable forms of treatment.

If success in the treatment of malignant tumors of the nose and sinuses is to be attained an early diagnosis is extremely important.⁴ One common and often early symptom is a dull persistent pain about the face or head which is



Fig 51—Squamous cell epithelioma of the left antrum. Destruction of the outer wall of the antrum may be noted.

intensified at night or on lying down. Although other conditions can produce a similar type of pain, the physician should be suspicious of malignant disease and should attempt to confirm or disprove its presence.

From 60 to 65 per cent of the malignant tumors involving the nose and sinuses are squamous cell epitheliomas (Fig 51). The remaining 35 to 40 per cent are divided essentially among adenocarcinomas (Fig 52), round cell sarcomas, myxosarcomas, fibrosarcomas (Fig 53), chondrosarcomas, osteosarcomas, lymphosarcomas, melanotic epitheliomas, hemangioendotheliomas, and finally plasmocytomas, which are a form of multiple myelomas. Such neoplasms vary greatly in degree of malignancy and we believe that Broders' classification, which grades these tumors on a basis of 1 to 4

is essential in the proper selection of the method of therapy. In general, low grade lesions which are radioresistant are best destroyed with surgical diathermy, while high grade tumors which are radiosensitive are best treated by irradiation.

of metastatic lesions. Old or debilitated patients often are unable to endure the postoperative reaction following radical surgical or radiation procedures. For such patients as well as for those who have hopelessly extensive tumors,



Fig 52—Adenocarcinoma of the antrum. A the expansile character of the tumor of the right antrum may be noted. B the adenocarcinoma is of the destructive type the loss of the outer wall of the right nostril and antrum may be noted.

tion. Secondary hemorrhages following the extensive use of diathermy or radium may be profuse, but usually can be controlled by the insertion of a tight gauze pack into the cavity. Only rarely does ligation of the external carotid artery become necessary.

treatment must be limited to palliative irradiation. Fortunately, malignant tumors of the nose and sinuses seldom metastasize to the cervical lymph nodes except late in the course of the disease. When the cervical lymph nodes have become involved to a limited degree and when the malignancy of the primary lesion is of low grade, block dissection of the cervical nodes is to be recommended. Metastatic lesions of extremely active growths are probably better treated by irradiation.

In most instances, treatment of malignant neoplasms of the nose and sinuses is best carried out under intratracheal anesthesia, which provides a satisfactory airway regardless of blood clot into the pharynx or of the time consumed in removing the tumor.

Malignant Tumors of the Nasal Cavities— Malignant tumors occurring in the nasal cavities produce nasal obstruction, discharge, and bleeding. They may vary in size from tiny lesions to growths which fill the entire nasal cavity, bulge into the nasopharynx, or invade one or more of the sinuses. Occasionally, extensions of the growth may produce olfactory and optic disturbances or the lacrimal sac may become involved to produce epiphora. Although these tumors may develop more rapidly than those of a benign nature, the signs and symptoms fre-



Fig 53—Fibrosarcoma grade 2 of the left antrum, malar bone and orbit.

In addition to the grade of activity, other factors must be considered which often alter the choice of treatment. Such factors are the age and physical condition of the patient, the extent of the lesion, and the presence or absence

quently are essentially the same as those produced by benign tumors. Consequently, the necessity of a microscopic examination of all nasal tumors should require no further emphasis. Many malignant tumors of the nose become ulcerated and infected. Consequently, if a rather superficial specimen is taken for biopsy, only inflammatory necrotic tissue may be found. A negative report, therefore, does not always rule out the presence of malignancy, a fact which cannot be stressed too strongly.

Malignant tumors may arise from any portion of the nasal cavity but are likely to originate on the septum or high on the lateral walls of the nasal passages. The fact that many malignant lesions of the nose are merely extensions of carcinomas which arise primarily in one of the accessory sinuses should not be overlooked. In the majority of cases, roentgenograms of the sinuses are essential. The roentgenographic finding of erosion of the bone which is produced by malignant tumors is well known.

The choice of treatment for malignant tumors of the nasal cavity is based on the location, extent, and degree of activity of the lesion. Highly active malignant neoplasms, particularly neoplasms of grade 4, are treated most satisfactorily by the insertion of radium needles or radon seeds directly into the tumor mass, regardless of the size and location of the lesion. After such implantation of seeds, external irradiation also should be employed. Lesions of low grade are best treated by thorough electrocoagulation. If the limits of a low grade tumor are ill defined, the implantation of radon seeds through the involved region following electrocoagulation of the visible portion of the growth is to be recommended. In many instances, lateral rhinotomy is helpful in gaining exposure of the affected nasal cavity.

Frequently encountered in the floor of the nose are carcinomas of low grade which are difficult to visualize. In such situations a transantral approach to the tumor provides the necessary exposure for its removal with surgical diathermy.

Malignant tumors high on the antral wall of the nasal cavity and those of low grade of malignancy in particular are best treated in a manner similar to that which will be described for malignant neoplasms of the ethmoid region.

We would like to stress the importance of microscopic examination of lesions involving the anterior part of the septum. Not infre-

quently, ulcers in this location appear benign clinically but on histologic examination are found to be hemangio-endotheliomas. Even hemangio-endotheliomas of low grade are highly radiosensitive and after their destruction with surgical diathermy the implantation of radon seeds or the local application of a tube of radium is advisable.

Malignant Tumors of the Ethmoid Region — Malignant tumors in the ethmoid region may arise primarily in the ethmoid cells or may occur as secondary extensions from lesions arising in the nasal cavity. Growths arising in the ethmoid cells soon invade the nasal cavity and produce all the symptoms associated with a malignant tumor of the nose. Later in the course of the disease, extensions into the orbit with displacement of the globe and involvement of the orbital nerves as well as the olfactory nerves are to be expected. If a malignant tumor of the ethmoid region is extremely malignant, the insertion of radon seeds through the nostril probably will accomplish as much as any other form of treatment. However, in lower grade tumors, a combination of electrocoagulation and local irradiation becomes necessary. Under such circumstances, exposure of the ethmoid region is the difficult problem. Two methods of gaining access to the ethmoid cells are to be recommended: (1) The transantral approach through the mouth and antrum on the affected side requires the removal of a large portion of the mesial wall of the antrum and thereby provides direct visual access to the ethmoid region. (2) A fronto-ethmoid approach to the ethmoid cells has been used by Havens, Holmgren, and Moore with considerable success. This method of exposure of the ethmoid region involves an incision through the inner extremity of the eyebrow on the involved side. This incision is carried down along the side of the nose. Access to the nasal cavity subsequently is gained by removal of a portion of nasal bone and the nasal processes of the maxillary and frontal bones. Finally, the ethmoid cells are exposed by removing the lacrimal bone and a portion of the lamina papyracea of the ethmoid bone.

Regardless of which approach to the ethmoid bone is employed, the tumor itself is thoroughly electrocoagulated down to its attachment in the bony structures of the ethmoid region. This is followed by the accurate implantation of radon seeds or by the application of radium needles or a tube of radium directly at the point of at-

tachment of the tumor. The radium is held in position by gauze which can be removed through the nostril.

Considerable reaction may follow the use of surgical diathermy and radium about the ethmoid region. In some cases, edema of the brain ensues, but this condition usually clears up in a few days. Meningitis is a rare complication which is less likely to occur if the growth is removed by diathermy than if other surgical measures are employed.

Malignant Tumors of the Maxillary Sinuses — Because of their location within a bony cavity, primary malignant tumors of the maxillary sinus may become large before they produce any symptoms except pain. Eventually, however, erosion of the antral wall occurs at one or more points, this erosion permits extension of the

mus of the jaws. If the lacrimal sac becomes obstructed or involved by the malignant process, epiphora may be a conspicuous symptom.

The possibility that a malignant mass in the antrum may not be primary in this region should not be overlooked. It may be in reality an extension of a carcinoma which originates in the nose or upper jaw.

In the diagnosis of malignant tumors of the antrums, not only is an evaluation of the signs and symptoms of the disease important but the procurement of roentgenograms is indispensable. In most instances, roentgenograms of the involved antrum will show evidence of erosion or actual destruction of bone and the tumor itself will be revealed as a definite area of increased density. In the differential diagnosis, inflammatory lesions, benign tumors, cysts, and various tumors of the jaws, such as osteitis fibrosa cystica, giant cell tumors (Fig. 54), and adamantinomas, must be considered. In case of doubt as to the nature of the growth, conservative treatment is to be discouraged. On the contrary, the antrum should be explored by means of a Caldwell-Luc operation and tissue obtained for histologic examination by the fresh frozen section method.

Because malignant lesions of the antrum seldom are confined to the anatomic limits of the maxilla, we are of the opinion that resection of half the maxilla for the removal of such tumors is of little value. We believe that the most satisfactory results can be obtained by the use of surgical diathermy or irradiation or both; the choice of treatment is dependent on the location and activity of the growth. Regardless of which one of these methods of treatment is employed, it is advisable to destroy by electrocoagulation a portion of the anterior wall of the antrum, the alveolar process, or, occasionally, a portion of the adjacent hard palate, depending on where the tumor presents into the mouth. This procedure provides a permanent and adequate opening into the antrum for exposure at the time of operation and for subsequent inspections of the antral cavity in the months to follow.

In general, low grade malignant lesions should be destroyed with surgical diathermy. This procedure is not always easy. It is, of course, important to destroy all the growth no matter to what region it may extend. In more active lesions, we recommend that all of the visible growth be destroyed with surgical diathermy after which



Fig. 54 — Giant-cell tumor involving the floor and the outer two-thirds of the left antrum.

growth to structures external to the sinus. Extension in this way may produce a growth in the nasal cavity which has all the signs and symptoms of a primary carcinoma of the nose. Downward extensions of malignant tumors of the maxillary sinuses through the alveolar process cause loosening of the upper posterior teeth which is often one of the first symptoms of malignant disease of the maxillary sinus. Bulging and erosion of the palate also may occur. If the tumor infiltrates upward, the eye may become proptosed or displaced, such involvement of the orbital contents may produce fixation of the eyeball and blindness. Not infrequently, the neoplasm bulges or even infiltrates the cheek or may pass backward to involve the pterygoid region with a resultant tris-

three or four tubes containing 50 mg. of radium each are inserted into the postoperative cavity. A total of 1000 to 1400 mg. hours of radium is usually given and this treatment is followed later by external irradiation. If the growth is extremely active and radiosensitive, there is no advantage in destroying the growth with diathermy, as much can be accomplished by insertion of tubes of radium directly into the tumor. As might be expected, patients who have extremely malignant antral growths are more likely to be cured than those who have low grade malignant neoplasms for the radiosensitive tumors of high grade of malignancy can be destroyed by intensive irradiation whereas it is often technically difficult to destroy every particle of a low grade malignant tumor with surgical diathermy. Moreover, the patients whose malignant tumors are confined to the upper and posterior part of the antrum are more difficult to cure than those whose growths are in the lower and anterior portions of the sinus, because the former tumors are more inaccessible.

For patients who are extremely poor surgical risks, who are very old, or who have extensive cervical metastatic lesions, radical surgical procedures are unwarranted. Instead, palliative irradiation is to be recommended.

About 10 per cent of patients who have malignant tumors of the antrum lose the eye on the involved side after operation. This result is unavoidable and is due to injury from the treatment itself or to secondary ophthalmitis. Unless the eyeball itself or the tissues immediately adjacent are involved by the tumor, it usually is advisable to defer enucleation of the eye until the fact that it is hopelessly damaged is established.

The end results in the treatment of malignant tumors of the antrum have been reported previously. There is no field of malignancy in which there has been such marked improvement in the end results as there has been in the treatment of these tumors.^{5,6}

Following successful treatment of a malignant neoplasm of the antrum, the resultant perforation into the upper jaw is best closed by insertion of a dental prosthesis. External perforations and defects require plastic repair, but not until it is certain that recurrence will not develop.

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TRAUMATIC MAXILLARY SURGERY

Facial fractures are apt to be compounded either to the outside, or in to the mouth, nose, or sinuses. The term "compound facial injury" is thus used with the understanding that the predominant injury may be of soft tissue or bone.

Concomitant injuries are common and include skull fractures, concussion of the brain, fractures of the cervical vertebrae, and ocular injuries. Fractured and loosened teeth are almost the rule rather than the exception.

Preliminary Examination.—The occlusion of the teeth is important in establishing the presence or absence of fractures of either jaw and the solidity of the tooth-bearing portions may be determined by gentle manipulation. Pain in front of the ear upon opening the mouth, with deviation of the lower jaw toward that side, usually indicates a condyle fracture, if the entire lower jaw is displaced directly backwards, fractures of both condyles may be present. Difficulty in opening or closing the mouth without deviation may be due to impingement of a fractured zygomatic arch on the coronoid process, or simply to fractures in other portions of the lower jaw.

The rims of the orbits are palpated, especially the orbital floors, the patient is examined for diplopia, and rough estimates of visual acuity may be made. The extra-ocular movements, fundi, corneas, and anterior chambers are ex-

amined and any subconjunctival hemorrhage is noted

The anterior walls of the maxillary sinuses the zygomas, and the zygomatic arches are palpated. Crepitant air in the cheeks usually means fractures involving the sinuses although it may follow ordinary nasal fractures if the patient forcibly exhales against blood clots or nasal packs

Any blood is cleaned out of the nose and the interior examined for any mucosal tears. The

bits and the walls of the sinuses may be determined as well as the presence or absence of blood in the latter. Some information regarding the nose may be obtained and this may be supplemented by a lateral flash exposure made on a small dental film held against the side of the nose. A basal view with the tube beneath the chin and the film above the head, may show the zygomatic arches.

When to Operate upon the Patient—Facial and jaw fractures are preferably reduced early

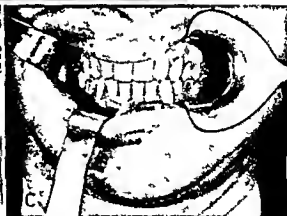
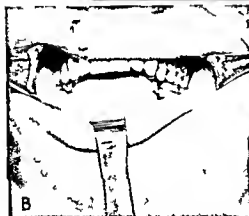


Fig. 55—Sample interdental wiring. Twelve-inch lengths of #24 stainless steel wire are looped around each of the bicusps and tightly twisted. Opposing wires are then twisted together, cut short, and the ends bent inward flat against the teeth. A, B, cusps are best for wiring; molars next, and canines and incisors poorest, but there should be at least one set of wires anterior and posterior to the fractures (B and C).

condition of the airways and the septum is noted and the nasal bones are palpated.

Roentgenograms of the lower jaw should include lateral views of both sides and a postero-anterior view. Lateral laminagrams often delineate the condyle and joint better than plain films, 2½ cm usually being the best depth. Roentgenograms of the facial bones are less helpful because of superimposition, but an ordinary Water's film without the cone is usually exposed. The continuity of the rims of the or-

bits and the walls of the sinuses may be determined as well as the presence or absence of blood in the latter. Some information regarding the nose may be obtained and this may be supplemented by a lateral flash exposure made on a small dental film held against the side of the nose. A basal view with the tube beneath the chin and the film above the head, may show the zygomatic arches.

Treatment of shock, cranio-cerebral injuries, or other serious lesions may take precedence, but rarely justifies delays of weeks or months. An

intoxicated or nauseated patient should not have his jaws wired together

Anesthesia—Local anesthesia induced with novocaine is quite generally relied on, but is often supplemented by a basal anesthetic. Children or nervous adults may require a general anesthetic, usually administered endotracheally (or by endopharyngeal insufflation in infants). Intravenous anesthetics require constant attention to the airways and may be quite troublesome in these patients.

Deep blocks of the second or third divisions of the trigeminal nerve are commonly employed. Infra-orbital, supra orbital, mental, mandibular, and superior dental nerve injections all have their own special fields of usefulness. Local infiltration of wounds and topical application to mucosal surfaces will suffice in some instances.

The nasal and oral airways are then cleared, if necessary, and hemostasis of all wounds secured. Following this, a more adequate examination may be possible and additional notes are made as to the injuries, parts totally missing, and other observations, and a plan of repair is formulated.

The fractures are ordinarily reduced first, followed by fresh cleansing and changing of drapes, and any other procedure or procedures deemed necessary, after which the soft tissues are closed, another set of instruments being utilized.

Methods of Fixation for Jaw Fractures—Interdental wiring is the most generally used and will suffice for most ordinary fractures when a sufficient number of solid teeth are present. There are many types of wiring, but the most simple one is the Gilmer type (Fig. 55).

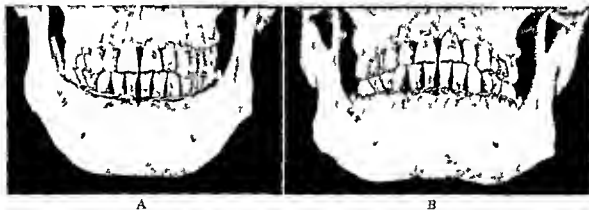


Fig. 56—A Heavy, half round steel wire used as a labial arch bar. It is individually ligated to most of the teeth in the lower jaw with fine wires and then regular interdental wiring between the two jaws. B Risdon's arch bar. A length of #24 stainless steel wire is looped around the back lower molar tooth on each side and twisted out for several inches. The two are then twisted together in the midline anteriorly to form a twisted wire arch bar. Ligation to individual teeth with #26 or #28 wire and usual fixation to upper jaw.

General Plan of Operation—The skin surfaces are gently cleansed with soap and water, with the addition of ether or benzene if any oil or grease is present. It is particularly important to remove the latter, as well as any other dirt or cinders, from all abrasions and lacerations to prevent subsequent tattooing. All lacerations are copiously irrigated with saline, after which any mild antiseptic of choice (preferably one that does not stain or discolor) is applied to the skin and sterile draping is done. The question of when to cleanse and when to anesthetize is sometimes difficult to decide, but ordinarily cleansing is done first until the patient becomes too uncomfortable, after which cleansing and anesthetizing are done alternately until complete.

Dental arch bars are anchored with fine wires to the labial surfaces of individual teeth throughout the arch for some fractures. Any of the manufactured arch bars or Risdon's method may be used (Fig. 56).

Internal wire fixation is often the most simple and direct method of fixation for edentulous jaws or fragments (Fig. 57). It consists of manually reducing the fracture and while holding it in place, drilling one or more Kirschner wires through the bone and across the fracture site. The Kirschner wires should avoid the nerve canal and any tooth roots and are put in with sterile precautions, preferably by aid of a power drill.

External bar fixation is obtained by drilling pins or screws perpendicularly into each of the

fragments from the outside and then anchoring them with individual lugs to an external rigid metal bar (Fig 58)

Direct bone wiring by open operation presents certain hazards and is avoided whenever possible, but is occasionally used. *Circumferential wiring* of the bone fragments in oblique fractures is done at times, as is *circumferential*

of the teeth and the patient's sensations may be valuable guides as to the exactness of the reduction

Fractures of the body can usually be immobilized by simple Gilmer interdental wiring to the upper jaw if a nearly normal complement of sound teeth is present. The bicusps are best, the molars next, and the incisors and canines



Fig 57—Internal wire fixation. A Bilateral fracture in a totally edentulous jaw. The patient was able to put in his upper and lower dental plates and eat soft foods after the first week, though the lower plate was painful if left in continuously. He returned to work in two and one-half weeks. B Angle and mental foramen fractures in an almost edentulous jaw. Open reduction was avoided. C and D Fixation of an almost edentulous jaw in an insane man, which was solid enough to hold without any cooperation. (From Brown J B., and McDowell F. Internal Wire Fixation of Jaw Fractures—Second Report. Surg. Gynec. & Obst. 75:361, 1942.)

wiring of edentulous jaws to dentures or splints but often leaves much to be desired.

Plaster of paris head-cap appliances and dental splints are used as indicated in some instances but are not employed routinely.

Treatment of Lower Jaw Fractures—Where possible, immediate manual reduction is preferred to gradual elastic traction. The occlusion

poorest for the interdental wiring. Both sides of the jaw should be wired for any fracture, and there should be one set of wires anterior and one set posterior to each fracture, if possible.

Symphysis fractures require a dental arch bar in addition to interdental wiring to the upper jaw, to prevent rocking motion in the fracture line.

Condyle fractures usually require only fixation of the teeth in occlusion, preferably by interdental wiring. Most fractures of the coronoid

rior fragment. If their obliquity is such that they tend to lock in place when the anterior fragment is fixed by interdental wiring, that may

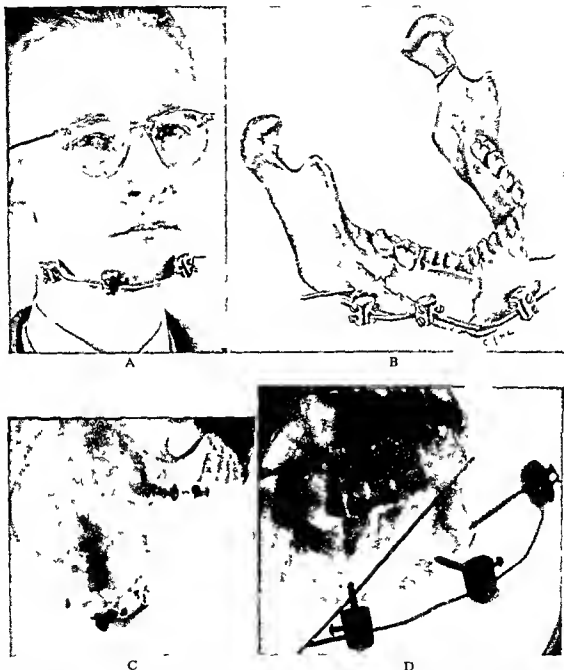


Fig 58—External bar fixation. Shattering jaw injury with partial collapse of airway. The patient arrived sitting in an ambulance with accompanying physician holding his jaw forward when necessary. A large fragment had been displaced down into the soft tissues of the neck. All the major fragments were saved, reduced, and immobilized with external bar and internal wire fixation. A few interdental wires were also placed where teeth were present, because of condyle fracture. Solid union with good occlusion was obtained. Late bone grafting was avoided. Lugs were designed by Dr James A. Brown (From Brown, J. B., and McDowell, F. Internal Wire Fixation of Jaw Fractures—Second Report, Surg., Gynec. & Obst. 75:361, 1942.)

or upper ramus may be treated in the same manner.

Angle fractures are particularly prone to non-union or malunion with elevation of the poste-

rior fragment. Otherwise, the interdental wiring can be supplemented by internal wire fixation, or by direct wiring, or by external bar fixation.

Edentulous jaws or fragments may be immobilized by (1) internal wire fixation, (2) external bar fixation, (3) direct wiring of the bone fragments, (4) circumferential wiring of the bone fragments to an intact denture or to a splint, or

Drainage of the fracture site through a small dependent external incision is usually the safest course, but may be modified at times by local or general chemotherapy, particularly if there is no displacement of the fracture.

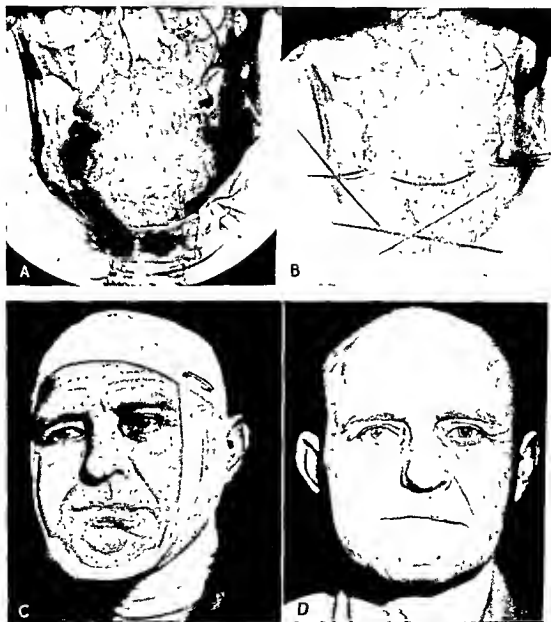


Fig. 59—Multiple fractures of the lower jaw and transverse separation of the upper jaw. The main fragments of the lower jaw were held with internal wires drilled across the fracture lines. B, Second plane of fixation with dental arch bar, which was also used to secure the alveolar block containing the four lower incisor teeth. Interdental wiring to upper jaw and use of closing muscles and elastic Barton bandage (C) to support upper jaw. D, Solid union of all fractures with normal appearance of face.

(5) circumferential wiring of the bone fragments to each other (in an oblique fracture). When just a crack is present without any displacement, a Barton's head bandage with the dentures kept in the mouth may suffice.

Length of period of fixation varies from three weeks for fractures of the condyle, coronoid, or upper ramus, to six weeks for fractures of the body. Badly comminuted or infected fractures may require even longer fixation and the diag-



Fig 60.—Depressed orbital floor associated with nasal fracture A Typical appearance after injury The depression in the floor could be palpated and the patient had diplopia when the eyelids were held open The floor was elevated through the antrum and the nose was brought forward and held by silver wire mattress sutures behind it (secured over lead plates) B Final appearance with eye on good level The patient has binocular vision



Fig 61—Zygomatic arch fracture A Impingement on coronoid prevented free opening and closing of the mouth Scalp incision was made in the temporal region down through the temporalis fascia An elevator was passed beneath temporalis fascia and under the arch to pry it out into place with result shown in B

nosis of nonunion is not made until after twelve weeks of continuous immobilization. Union is determined clinically, and not by roentgen ray

Teeth in the fracture lines are not removed immediately in every case, particularly if they assist in the fixation in any way. This is espe



Fig 62 —Lateral displacement of nose. Fresh fracture. Easily reduced by moving it over to the midline and holding it there with an external aluminum splint.



A

B

Fig 63 —Aluminum nasal splints. These are cut out of sheets of 250 aluminum alloy 0.0016 inch thick and are covered with mushlin held on with rubber cement. The side walls are bent in and shaped to the nose to hold the nasal bones inward. Splint anchored to forehead and face with adhesive strips.

study, as fracture lines may persist on the films after the jaw is subjectively and objectively solid

cially true of any teeth in the posterior fragment of a molar fracture. If a low grade infection develops in the fracture line it may be neces

sary to remove the tooth before solid union is obtained, but serious infections due to this cause are rare if external drainage is employed

Nourishment is maintained by a high caloric liquid diet at first, with the later addition of any puréed or ground foods. There is enough room behind the molar teeth for these foods and it is not necessary to extract any anterior teeth for feeding. Attention is paid to all of the nutritional requirements and the weight should remain nearly constant during the period of fixation



Depressed orbital floors are usually reduced through the antrum. An incision is made in the canine fossa, the antrum entered through one of the fracture lines, the floor pushed up with a blunt elevator, and the antrum packed with iodoform gauze which is left in for about one week (Fig. 60)

Zygomatic Fractures—Zygomatic arch fractures tend to impinge on the coronoid and can be reduced by Gillies' method. A small incision is made in the temple and carried through the temporalis fascia. An elevator is then passed



Fig. 64—Depressed fracture of the nose. Blunt elevator introduced through the nostrils to bring the whole nose forward and take the buckles out of the septum. Bones squeezed inward and held inward and forward with external aluminum splint. Torn mucosa lightly packed in place

Treatment of Upper Jaw Fractures—*Transverse fractures* just above the alveolus tend to hang down and need only to be held up in place with the teeth in proper occlusion. This is usually accomplished by interdental wiring reinforced by an elastic bandage wrapped tightly over the vertex and under the chin (Fig. 59). The powerful closing muscles of the lower jaw help in keeping the fragment up in place. A Risdon arch bar on the upper teeth may help when only part of the upper jaw is separated.

Vertical fractures are managed in much the same way, but are apt to have concomitant fractures of the zygoma or orbital floor also requiring treatment.

downward underneath the fascia and the arch is pried outward into place, usually locking (Fig. 61). Direct elevation with a single tenaculum applied locally can also be performed.

Fractures of the zygoma may present more difficulty. Some may be reduced by Gillies' method. Others can be approached best through the mouth, particularly if the orbital floor is depressed. If the zygoma is torn loose from all of its mooring, it may be best to do a direct drilling and wiring at the external angular process and to pack the orbital floor up in place through the mouth.

Nasal Fractures—Lateral deviations are pushed back to the midline and held in place

for ten days with an external aluminum splint (Figs 62, 63)

Depressed fractures are elevated up out of the face with any blunt instrument (e.g. a Kelly clamp with the blades covered with rubber tubing) introduced through the nostrils. This

ternal aluminum splint (Fig. 64) Packing is introduced back along the floor of the nasal airways toward the pharynx and is used to maintain the airways and to hold any torn mucosa flaps in place. It is not used to hold the nasal bones forward.



Fig. 65—Avulsion of nose without fracture. The patient was injured 200 miles away but a local doctor gave best possible first aid treatment (*i.e.* replacing the flap and wrapping tight pressure bandage around entire upper face). When the patient arrived the flap was viable and it was cleansed and sutured in place with multiple tiny 000 black silk stitches. Good hemostasis was obtained and pressure dressings applied with the addition of an external aluminum splint to maintain the shape of the nose. B and C: Appearance four days later.

maneuver should also take any fresh buckles out of the septum and one should be sure that the airways are restored. The nasal bones are squeezed inward (in any depressed fracture the nasal bones are flared outward at the face) and held in, either with a large wire mattress stay suture put in over lead plates or with an ex-

ternal aluminum splint (Fig. 64). Packing is introduced back along the floor of the nasal airways toward the pharynx and is used to maintain the airways and to hold any torn mucosa flaps in place. It is not used to hold the nasal bones forward.

Soft Tissue Injuries—Debridement is conservative as possible, relying mainly on cleansing and preferring to leave in any tissue that might die rather than removing any that might live but smoothing any small ragged edges.

Suturing is done almost entirely with 000 waxed black silk on fine curved cutting needles.

putting the stitches in very close to the wound margin, and relying on many minute stitches rather than a few coarse ones (Fig 65). Known points, such as the vermillion borders, edges of a nostril or an eyelid, or portions of a brow, are put together first. Intervening portions of the wound are closed by successively bisecting the remaining gaps. Interrupted subcuticular 000 white sutures are used to take the tension off of the surface stitches. Stay sutures are avoided when possible, otherwise put in through the mouth, or if necessary put in through the face and tied over a pad of gauze to obviate ladder marks across the scar.

Through and through lacerations are closed both on the mucosal and skin surfaces, with a few 000 white sutures in the intervening layers when advisable. The possibility of a torn or lacerated parotid duct is considered.

Pressure dressings with mechanics waste, gauze rolls, bandage, and adhesive are routine and do much to prevent edema and keep blood clots and serum out of the wounds. A single layer of fine mesh grease gauze over the suture lines will keep the dressings from adhering to the stitches. Drains are seldom necessary if adequate pressure is obtained, but small rubber bands may be used when desirable.

Secondary plastic procedures may be necessary in any extensive injury and should be discussed with the patient or his relatives at the start. They will be lessened in amount and easier to execute if a careful primary repair is done soon after the injury.

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RECONSTRUCTIVE SURGERY OF THE NOSE AND EAR

General Considerations—Plan of Reconstruction—The amount and type of tissue lost—covering skin, supporting tissue (cartilage and/or bone), and lining membrane—must be accurately determined. It is not sufficient to make the estimates from the healed, contracted defect. This is true particularly in losses due to burns, infections, and trauma with secondary infection and consequent marked contractions.

In reconstructive surgery of the nose, an epithelial lining must be provided in all cases of full thickness loss of the nasal wall and in some instances where the loss involves lining only. Skin, which matches or closely approximates the color and quality of nasal skin, should be selected for surface losses. Losses of supporting structure (bone and/or cartilage) may be replaced by either or both of these tissues or with a soft tissue implant (dermal graft). The object in reconstruction is the restoration to normal or as near the normal as possible. This applies to both function and cosmesis.

Procedures of choice are those which produce the nearest approach to normal function and appearance without creation of new cosmetic disabilities in the neighborhood or at a distance. Lining is provided by utilizing bordering covering tissues or by sliding lining downward from beneath the bony arch. A nasal surface defect is repaired with a free full thickness graft removed from the mesial surface of the ear. This graft may be removed later, in many instances, by "multiple excision."

Procedures of necessity are those which must be employed when the loss is of such extent or character that a repair confined to the site of the injury is impossible. They utilize interpolated flaps of skin, which are advanced or rotated on a pedicle, from neighboring regions (forehead and/or face and/or lip) or from a distance, the flaps being transferred on a tubed pedicle or carrier (such as an arm or hand). Free flaps, either full thickness or "split" portions, are frequently employed. At the present time, the use of pedicled flaps from a distance and free flaps (grafts) is a common practice. They never produce a pleasing cosmetic result because the color and texture contrast becomes the most prominent feature of the face.

Skin Flaps—THE FRENCH OR SLIDING FLAPS—This flap has a limited use in covering a surface loss. It is slid or advanced from its original bed with slight twisting or torsion of its pedicle. It must never be used in the repair of full-thickness losses without provision of lining tissues.

INTERPOLATED FLAPS—These flaps are required for the repair of all major losses. Several methods of obtaining tissue for interpolation may be defined.

1. **Flaps from Immediate Neighborhood (Indian Mutilation Type)**—Such flaps are single or double with a pedicle which is rotated,

twisted, bridged, or tunneled. They are the ones of choice for large partial and total nasal covering (See Fig 79)

2 Flaps from a Distance with Tubed Pedicle—"Tubing of a pedicle" consists in suturing the raw edges of a rectangular flap, thus doing away with the danger of infection by including raw surface and furnishing certain blood supply to a flap at its distal end. The base of this pedicle is so located that the distal flap can be transferred without undue twisting or tension of the pedicle (Fig 66)

3 Flaps Transferred on a Carrier—This is essentially the Italian method which was primarily proposed for rhinoplasty. It consisted in fashioning a flap on the arm and later transferring it to the nose with the arm immobilized

Tubes may be fashioned elsewhere on the body and either swung or advanced caterpillar fashion to the defect

4 Delayed Flaps (Perthes' Method)—Flaps of either the sliding or interpolated type must not be removed from their origin until an adequate blood supply has been assured. This applies particularly to interpolated flaps, which, of necessity, are cut, often, against or across the direction of the blood supply. They must be partially or totally raised from the surrounding tissue at intervals of two or three weeks until the blood supply is adequate, when the pedicle is turned or twisted, as required

FREE FLAPS OR GRAFTS—These are Thiersch grafts, split portions of skin, full thickness grafts, pinch grafts



Fig 66—Tubed pedicle

on the head until a new blood supply was established

In the light of present knowledge, the original method has nothing to recommend it and many things to condemn it. The position is torture to the patient. There is danger of emboli in the superficial veins, infection occurs readily from contact with the nose, and the dressings are difficult

The method, with proper modification of the position of the arm and management of the flap, is, however, the one of choice in the majority of instances in which such a plan of procedure is demanded

Flaps may be tubed at a distance (abdomen), attached to the hand or lower forearm, and transferred with the arm in a comfortable, safe position (Fig 67)

FLAPS OF CHOICE—The use of the forehead flap for minor and small full thickness losses is never warranted

Defects about the tip, alae, and lateral surfaces of the nose and moderate sized losses anywhere may be repaired by turning lining flaps from the border of the defect and covering the surface with free full thickness grafts

Losses in the upper half of the nose may be repaired with island flaps after the technic of Mook (see Fig 78) using the frontalis vessels as a pedicle

Large partial or total losses are best repaired with a forehead scalp-flap as a covering. This flap may be grafted to provide the lining or the lining may be turned in from the borders of the defect. The forehead flap may be used as both

lining and covering in some instances (luteic nose)

Lining Flaps—Lining tissue for the nose may be supplied by skin introduced either as a hinged, rotated, tunneled, or pedicle flap or as a free graft, variously applied. A free graft may be applied to the covering flap before it is transferred from its origin or on a temporary prosthesis, which is either sutured in position or retained mechanically by a device or a dressing (See Fig 76.) Examples of these uses will be exhibited in the discussion of various reconstructions

Implants (Supporting)—Some operators prefer bone or bone and cartilage for restoration of the nasal ridge. Most of the others choose costal cartilage, which is either autogenous or

the ribs and tibia does not offer the desirable quality of the cancellous bone of the iliac crest. Bone from the iliac crest is obtained with less discomfort and shorter disability than bone procured from sections of the rib and tibia. It can be readily fashioned in situ and transplanted with minimum handling.

Application of Bone Grafts to the Nose—Approach should be made through an incision in the nostril. The skin covering should be separated by sharp dissection and the periosteum elevated. By chiseling, the residual nasal bone and nasal articular surface of the frontal bone should be freshened. The graft is then inserted and held in position with a splint.

CARTILAGE—Most operators consider cartilage the material of choice for nasal support. It does not unite with bone. It is encapsulated in scar and remains movable.



Fig 67—Tubed pedicle.



Fig 68—Dermal graft

homologous (preserved). Some prefer soft tissue (dermal graft), which changes into flexible scar tissue, for the restoration of all but the gross defects.

BONE—Free or block grafts are the only suitable ones for use in the nose. The outer table of the frontal bone is difficult or impossible to handle with exactness in connection with the forehead flap.

Block grafts are solid pieces of cortical or cancellous bone which are fashioned in situ to the desired shape with a chisel, saw, and drill, or removed and shaped in a sterile vise on a side table.

Sources of Bone—Homologous or heterogenous bone should not be used since the patient has an adequate supply. Block grafts can be obtained from ribs and long bones (tibia) and the crest of the ilium. The cortical bone of

Both autogenous and homologous cartilage is usable. Autogenous cartilage is obtained from the free and fixed ribs. Homologous cartilage is gathered from the same source in the morgue under aseptic conditions and preserved as follows:

- 1 All of the perichondrium is removed carefully.
- 2 The cartilage is placed in a solution consisting of 1 part aqueous merthiolate and 4 parts physiologic saline solution.
- 3 The solution is changed on the following day, again three days later, and, after that, once a week.
- 4 The cartilage is stored in small sterile jars in a cool place.

Cartilage may be readily carved to proper shape, introduced through the nostril or columella, and retained in various manners.

DERMAL GRAFT—This is a most useful transplant for correction of contour. It is easily obtained and transplanted. It can be cut to desired shape and placed in double layers. This procedure may be repeated as often as necessary to obtain a perfect restoration. The graft organizes into scar, which remains flexible. It is useful particularly in the lower half of the nose. The rare trouble encountered is occasioned by sebaceous cysts which form in undegenerated glands.

Application of Dermal Grafts to the Nose—Size and shape of the desired section are outlined on the abdomen. The skin is dissected with a sharp knife and all fat is removed. The tissue is stretched over a board and the epithelial surface shaved or scraped away.



Fig. 69—Nasal splint. Sheet metal (20 gauge) covered with adhesive tape.

The transplant is introduced through an incision in the nostril into a bed tunneled beneath the skin by either sharp or blunt dissection. It is drawn into position by means of sutures placed in the upper corners and threaded to straight needles (Fig. 68). Sufficient skin to overcorrect the deformity 20 per cent is implanted. The nose is dressed with an eyepad and a splint under moderate tension.

Other Procedures—**MULTIPLE EXCISION**—The principle of multiple, partial excisions consists in the gradual reduction of cutaneous deformities. The rationale of the procedure is based on the fact that the skin in early and middle life is elastic and that it rapidly regains normal relaxation and elasticity after being put on marked stretch or tension. This is less true

of the nasal covering than other skin surfaces, but it remains a very useful procedure here.

The maximal amount of defect which permits closure without undue tension is excised. This may be determined by stretching the normal bordering skin toward the lesion and overlapping it with the dissected defect. The borders are approximated. When ultimate relaxation has taken place the procedure is repeated until the maximum result is obtained.

Z. PLASTIC OPERATION—This is one of the most useful and satisfactory plastic procedures. It affords a simple method of correcting some contractures and of exchanging normal surrounding skin for a defective area. The latter is transferred to a position where multiple excision may be employed.

Preparation and Anesthesia—In reconstructive surgery of the nose the routine preparation satisfactory to the operator should be employed for both the surface skin and that in the vestibule.

Procaine (0.5 per cent) with added epinephrine hydrochloride (5 to 10 minims to the ounce) is used to either block or infiltrate



Within the nose topical application of cocaine (10 per cent) in epinephrine hydrochloride (1:2000) provides satisfactory anesthesia in all adult cases.

Splints and Dressings—Simple splints, producing moderate pressure and maintaining desired form during the early stage of healing, are all that is required (Fig. 69).

Sterilized "Scotch Tape" or fine-meshed gauze applied with thin collodion makes an excellent splint for the retention of shape and position of the tip and columella during healing and organization.

Complications—These consist of (1) Poor blood supply in the flaps—resulting in either cyanosis or ischaemia. This, generally, is due to mistakes in planning and preparation. (2) Hem-

atoma serum collections and/or necrosis resulting from poor hemostasis and faulty dressing (3) Infection

Reconstructive Rhinoplasty (Losses)—Columellar Loss (Fig 70)—Reconstruction is accomplished with a grafted hinged interpolated mucosal flap from the posterior surface of the upper lip

Alar Loss (Ulceration) (Fig 72)—The lining and nostril margin are provided by a hinged interpolated skin flap from the nasal border of the loss. A full thickness graft from the mesial surface of the ear provides the skin covering.

Alar Displacement (Birth Injury) (Fig 73)—The required elongation of lining is effected by the Kazanjian procedure. The skin is incised

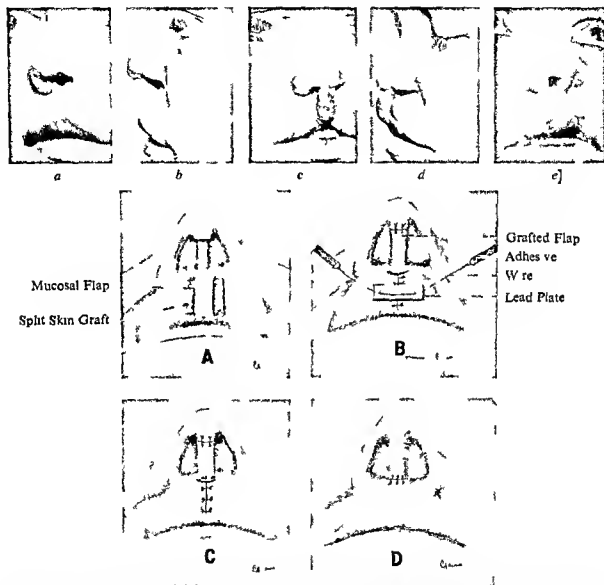


Fig 70—Columellar loss. *a* and *b* Appearance of patient before reconstruction. *A* incised borders of a rectangular flap of mucosa and underlying muscle. *c* and *d* split skin graft in position. *e* *B* superior end of mucosal flap sutured to the nasal tip. *C* lead plate and wire removed. *D* inferior end of columellar flap amputated from lip and sutured in position on the lip. *e* appearance of patient after reconstruction.

Alar Loss (Carcinoma) (Fig 71)—Reconstruction is effected with a hinged interpolated cheek flap for lining and nostril margin. The cheek defect is closed with a French or sliding flap. The nasal skin surface is provided by a free full thickness graft from the ear.

along the upper margin of the ala down to the lining and the mucoperiosteum freed from the nasal process. This is incised and brought down as indicated in the drawings. *A* rotated interpolated flap from the side of the nose and cheek provides the skin covering. A free full thickness

graft from the ear may be employed instead of this flap

Tip (Loss of Tip and Alar Attachment) (Fig 74)—Repair is accomplished with a hinged interpolated lining flap and a free full thickness skin graft from the ear

Tip and Columellar Loss (Fig 75)—The columella is provided by a grafted hinged inter

in place by a prosthesis which is supported by a cap splint fitted to the upper teeth. The columella is provided by a skin tube fashioned above the clavicle advanced to the cheek and finally sutured to the tip and lip in two stages

Atresia of the Nostril (Fig 77)—Reconstruction is effected in one procedure utilizing Z

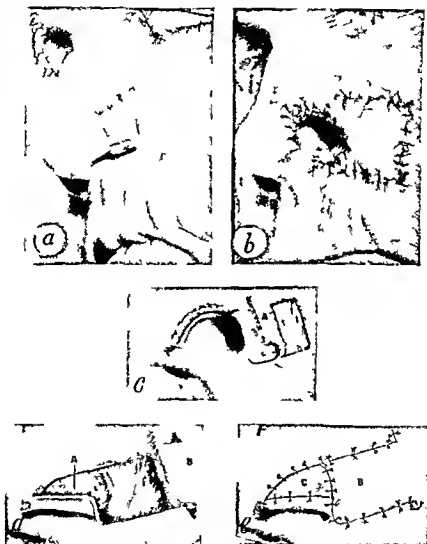


Fig 71—Alar loss (carcinoma) *a* Appearance following reconstruction *b* former appearance *c* hinged flap to form nasal lining and rolled alar edge *d* lining flap and alar border in position *A* covering flap *B* dissected *e* covering flap *B* sutured in position *free graft* *C* filling the alar skin defect

polated flap of nasal skin the consequent surface defect is covered by free full thickness graft of ear skin and this is finally removed by multiple excision

Columellar and Lining Loss (Carcinoma) (Fig 76)—Septum lower lateral cartilages lining and columella were removed with a carcinoma. New lining is provided by a split skin graft held

flaps. The usual method consists of dissecting off the contracted scar and covering the resultant raw surface with a split skin graft held in place by an obturator. This must be worn to control contracture for some weeks after organization of the graft.

Losses of Full Thickness of the Lateral Wall (Carcinoma) (Fig 78)—A pedicled forehead

flap (frontalis vessels) is elevated grafted with split skin and delayed until the graft is organized. The flap is drawn through a tunnel beneath the skin and sutured into the defect (Monk's method— island graft.)

Loss of the Glabella (Bird Beak Nose) (Fig 79)—A skin grafted delayed rotated inter

border of the defect. The skin covering is a full thickness graft from the ear. The saddle is corrected by a dermal graft which is planted in excess (20 per cent) to allow for subsequent organization.

Total Nose (Lupus) (Fig 81)—The lining may be provided by hinged flaps from the bor

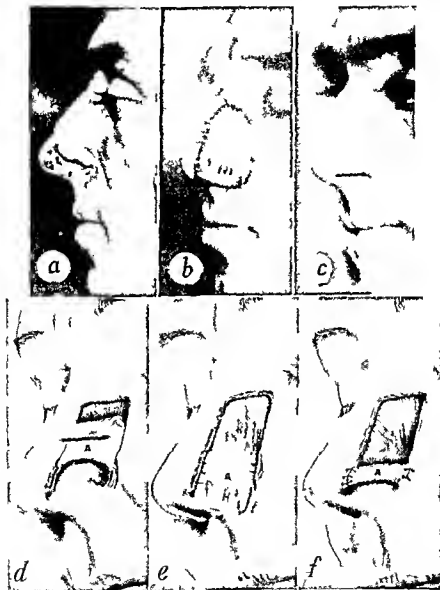


Fig. 72—Alar loss (ulceration) *a* Condition after the loss nasal covering skin sutured to lining of mucous membrane *d* hinged lining flap *A* dissected from lateral surface of nose *e* lining flap *A* sutured to nasal lining *f* lining flap *A* folded on itself and sutured to the skin bordering the defect to form the alar margin *b* Wolfe graft from mesial surface of ear to replace loss of nasal skin *c* appearance six months after operation

polated flap is used to repair the large defect. The small forehead defect is grafted with split skin after return of the pedicle.

Loss of Full Thickness of the Lower Half (Carcinoma) (Fig 80)—Lining is provided by a delayed hinged interpolated flap from the

unders by grafting the covering forehead flap before its transfer or by utilizing part of it for this purpose. The covering is provided by a rotated interpolated forehead flap (Indian). The support is bone and/or cartilage inserted after organization of the soft parts.

Burns (Fig 82)—In the case illustrated there has been total destruction of the nasal covering and distortion of the left ala following a third degree burn. The entire covering is supplied by full thickness skin from the mesial surfaces of both ears. The ear defects are grafted with split skin.

Cosmetic Rhinoplasty—The general principles of cosmetic rhinoplasty are well illustrated in the correction of a hump nose with a dependent tip and a deviated nose resulting from fracture.

Hump Nose (Fig 83)—The objectives of the operation—removal of the hump (nasal

3 The skin covering of the entire nose is elevated by sharp dissection (Fig 84 D)

4 Straight scissors are inserted with one blade beneath the skin and one in the nose and the upper lateral cartilage and lining are freed from the septum.

5 The nasal bone is sawed along the desired line on either side (Fig 85 A) this incision is carried through the cartilage with the knife (Fig 85 B) the free piece of bone and cartilage are removed (Fig 85 C)

6 The cancellous bone is chiseled out at the base of the cut in the glabellar region to allow proper narrowing (Fig 85 D)

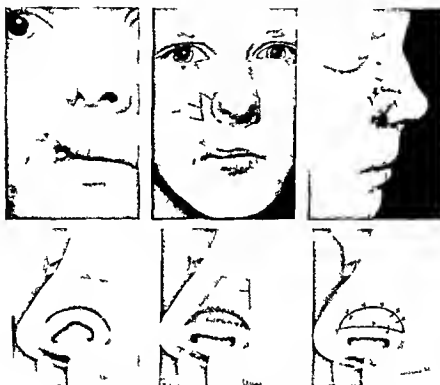


Fig 73—Alar displacement modified Kazanjian operation

bone and quadrilateral cartilage) to produce a straight or other desirable nasal ridge. Narrowing of the tip and elevation of the tip—are accomplished through the following procedures.

1 Incision is made through the lining skin in the nostril just below the upper lateral cartilage from the lateral attachment of the ala to the tip and on down the membranous septum to the nasal spine. This is repeated on the opposite side (Fig 84 A).

2 The periosteum is incised along the lower margin of nasal bone and nasal process of the maxilla. It is elevated in the required area (Fig 84 B C).

7 The lining is incised to the bone (nasal process) at the outer border of the glenoid fossa (Fig 86 A) the periosteum is elevated up to the frontal attachment in the groove formed by the nasal process and body of the maxilla (Fig 86 B) a saw-cut is made partly through the bone in this groove (Fig 86 C) a chisel is inserted between the nasal septum and previously cut nasal bone and the nasal bone and process are cracked outward. This leaves the mucoperiosteum intact. The nasal process may be severed from the maxilla with a chisel inserted intranasally or through the skin (Fig 86 D).

8 The sawed edge of the nasal bone and the septum are filed (Fig 87 A), the remnant of upper lateral cartilage is trimmed from the septum (Fig 87 B)

from the inferior border of the quadrilateral cartilage to permit the desired elevation of the tip (Fig 87 D)

10 A triangular shaped piece of skin and the

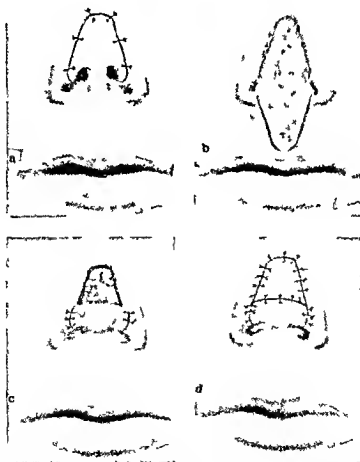
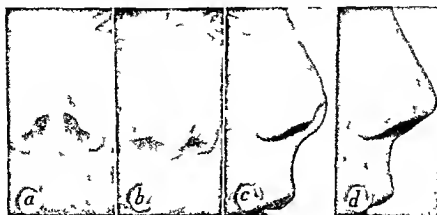


Fig 74—Loss of tip and alar attachments *a* and *c* Loss of nasal tip and tip attachments to the nasal alae, *b* and *d* appearance after reconstruction *a* Nasal skin flap to form lining of reconstructed ala and tip of the nose *b* flap reflected and sutured to the lining of the ala *c* flap folded and sutured to the skin bordering the defect of the ala to produce a redundant skin mass at the tip *d* full thickness graft filling the nasal defect

9 The columella is freed down to the nasal spine through the columellar incision (Fig 87 C), a proper wedged shaped piece is removed

lateral crus of the lower tip cartilage near the junction of the two crura are removed to permit narrowing of the tip (Fig 88 A)



Fig 75—Tip and columellar loss



Fig 76—Columellar and lining loss (carcinoma)



11 The mesial and inferior edges of the upper lateral cartilage are trimmed as required (Fig 88 B)

12 The columella is approximated to the septum with a few interrupted sutures. The nose is packed lightly with iodoform ribbon gauze. Splints and supports are applied externally as described (Fig 88 C-D)

ized by sawing, depending upon the shape of the ridge. They are not removed.

3 Deviated septum must be reconstructed either by (a) elevation of the mucoperichondrium on one side only freeing the cartilage from the vomer and perpendicular plate and the removal of sufficient properly located small strips (1 to 2 mm) of cartilage to permit the



Fig 78—Full thickness loss of the lateral wall (carcinoma) Monk's method— island graft

Deviated Nose (Fig 89)—Correction differs from that of the hump nose in three particulars

1 A long flat side results from the fracture and displacement. A wedge shaped piece of bone, having an angle equal to the angle of deviation of the nasal ridge from the center line, must be removed from the nasal process at its junction with the body of the maxilla.

2 The nasal bones may or may not be mobilized

by sawing, depending upon the shape of the ridge. They are not removed. (b) by partial submucous resection and sufficient cross cutting through the cartilage only at points of convexity to produce the above result.

Saddle Nose—This deformity may be congenital or may follow trauma or disease. In those cases in which there is no loss of lining, repair may be effected with bone and/or cartilage and/or dermal graft. Contracted lining scar must be removed and new epithelial lining

provided in cases where gross loss has occurred (such as in lues) before one of the above ridge supports can be employed successfully

In Figure 90 the left and middle pictures

In Figure 91 on the left is shown a saddle nose attributable to loss of bony support, on the right, its appearance after reconstruction with a dermal implant



Fig 79 Reconstruction of the glabella Forehead flap (Indian)



Fig 80—Full thickness loss of middle of nose

show a saddle nose resulting from loss of bony support The illustration on the right shows its appearance after reconstruction with implanted costal cartilage

A and B of Figure 92 show a saddle nose in which there is loss of the bony arch and mucosal lining and scar contraction The scar lining was replaced with split skin applied over a pros

thesis Costal cartilage gave support for the ridge and columella C and D depict the appearance after reconstruction

riorly and inferiorly) *a*, lateral view before and, *b*, after reconstruction, *c*, anterior view before and, *d*, after reconstruction

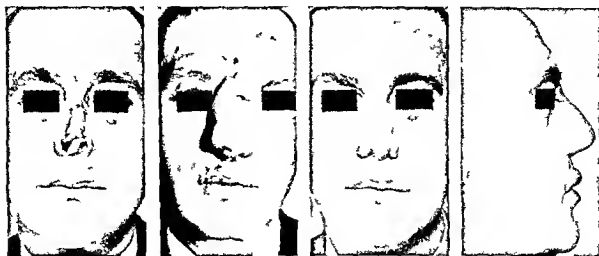


Fig 81 —Reconstruction of total nose



Fig 82 —Total skin covering (nose) *a* and *b* Scar and distortion of left ala following third degree burn, *c*, *d* and *e*, appearance of nose after removal of scar and after covering with full thickness skin from mesial surfaces of the ears

Figure 93 shows a saddle nose resulting from compound fracture of the nasal bones and nasal process of the maxillae (force exerted ante-

Congenital Anomaly —In Figure 94 the left and middle pictures show a congenital defect in which the free tube contains the lacrimal ducts

and sac. There is no nasal passage through the face on the right side. In the illustration on the right incomplete reconstruction is shown.



Fig 83—Hump nose

Otoplasty—Partial Losses Resulting from Disease Process or Trauma—HELIX LOSSES (Fig 95)—Helix losses which are small may be reconstructed from the borders of the loss. Larger losses are best corrected with a tube flap constructed immediately above the clavicle, advanced to the mastoid region, and later applied to the auricle. This tube should not be obtained from visible parts of the neck.

AURICLE DEFECTS (Fig 96)—These are repaired by the use of hinged and/or rotated interpolated flaps as illustrated.

AURICLE AND HELIX (Fig 97)—Partial losses are repaired by use of a sliding (French) flap of hairless skin from the mastoid region to provide the external surface and helix and a thick split graft to cover the created scalp defect. Large losses are better repaired by the methods of Kirkham¹ or Pierce³ (Figs 105, 106).

LOBULE (Figs 98, 99). This may be constructed satisfactorily by either of the two methods illustrated in the drawings.

CONGENITAL ANOMALY (Fig 100). On the left is illustrated a congenital closure of the external canal by an aberrant cartilage. There is malposition of a portion of lobule. On the right is shown the appearance after restoration of the canal. The aberrant cartilage was utilized to produce a tragus.

ATRESIA OF EAR CANAL (Fig 101)—The illustration on the left shows atresia of the ear

canal on the right. The appearance after correction by a Z-plastic operation.

ATRESIA OF THE EXTERNAL CANAL (Fig 102)—In a patient with atresia of the external canal repair was accomplished by removal of the scar and the insertion of a split skin graft over an obturator. The illustration shows the appearance several months after the repair.

HEMANGIOMA (CAVERNOUS) (Fig 103)—In cavernous hemangioma the cavernous mass is excised and the ear reconstructed.

Reconstruction of the Ear—TOTAL EAR—The total reconstruction of an ear is the most unsatisfactory procedure in plastic surgery. The qualities of the tissues involved and the contours required have seemed to preclude its successful accomplishment.

The use of the maternal ear cartilage has been disappointing because of its subsequent absorption in many instances and the fact that the covering skin is too thick to follow the cartilage contours. Only the general shape is obtained. The procedures of Kirkham and of Pierce (Figs 104, 105) may provide support (cartilage) which is more stable but suffer the

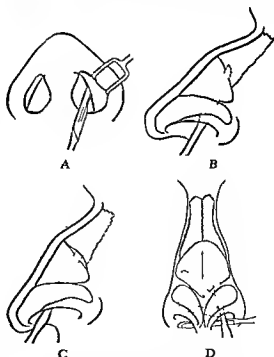


Fig 84

Figs 84-88—Nose drawings of operative technique for correction of hump nose.

same objections as to thickness, contour, etcetera. The methods are distinct advances, however.

SUBTOTAL LOSS OF THE AURICLE AND HELIX (Kirkham) (Fig 104)—Perforated, preserved

fashioned across the neck. The normal contour of the helix is outlined by incision through the skin covering the cartilage implant and this outlined skin flap is elevated with the attached car

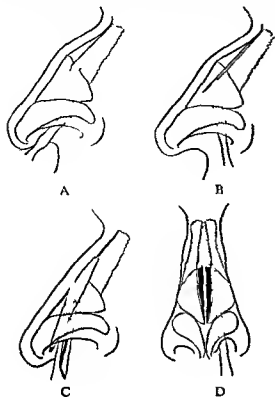


Fig 85

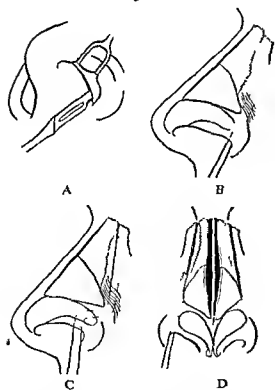


Fig 86

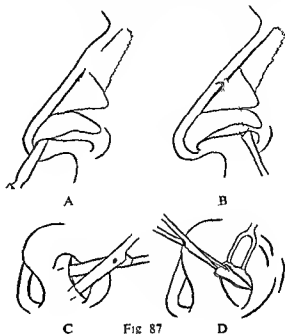


Fig 87

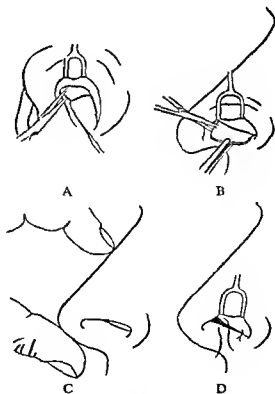


Fig 88

ear cartilage is implanted through an incision in the hair line in a position to replace the loss. A tube of skin based on the mastoid region is

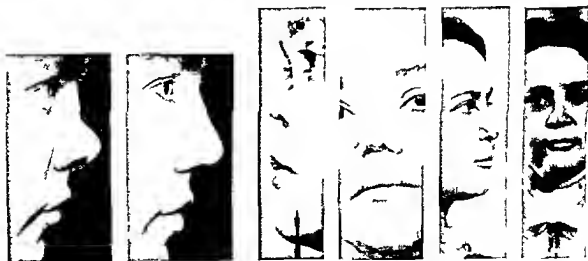
utilized. The raw surface on the mesial aspect of the ear is covered with a splint skin graft. The distal end of the tubed flap of skin is transplanted beneath the ear and the tube is ultimately arranged about the borders of the ear.



F g 89—Deviated nose



F g 90—Saddle nose bony arch Cartilage

Fig 91—Saddle nose bony arch
Dermal graftF g 92—Saddle nose (lues) Corrected in ng
Cartilage support

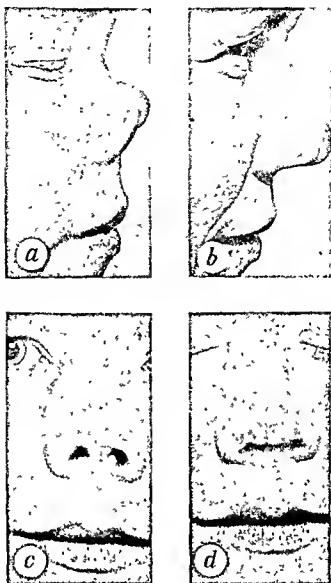


Fig. 93.—Saddle nose, old fracture Reconstruction.



Fig. 94.—Congenital deformity.



Fig 95 —Helix loss

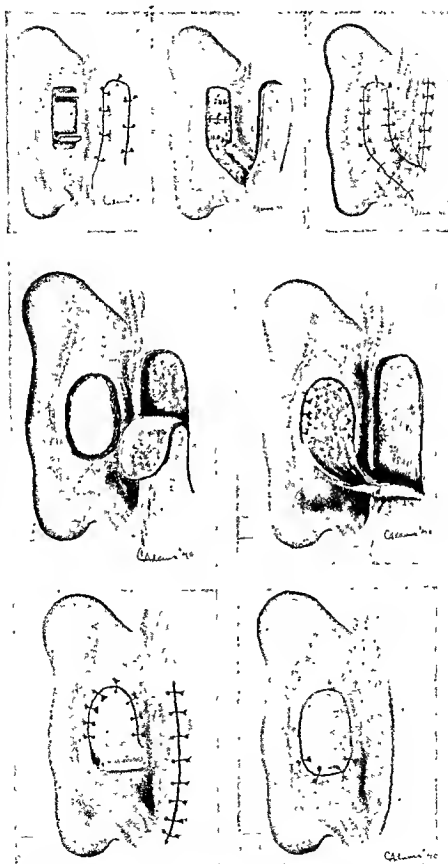


Fig. 96.—Auricle defect.

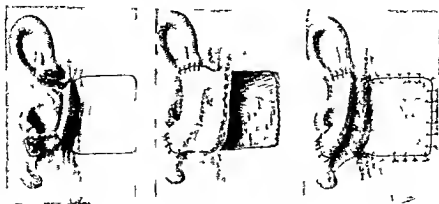


Fig 97—Auricle and helix

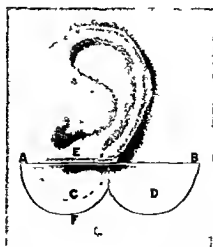


Fig 98—Ear flap (lobule loss) The straight line AB is incised along the border of the defect. Flap D and the posterior half of flap C are incised and elevated. The skin of the base AF is undermined. This flap is then returned to its bed. After an interval the line AB, the lower border of flap D, and the posterior half of flap C are again incised. Flap C and the tissue bordering the line AF are undermined. Flap D is folded onto the mesial surface of flap C and its upper edge, EB, is sutured to the skin bordering the defect on the mesial surface of the ear. The superior edge of flap C (AE) is sutured to the skin margin of the defect on the external surface of the ear. After an interval of three weeks the line AFE is incised. The lobule flaps are reduced to the desired size and their margins approximated with interrupted horsehair sutures.

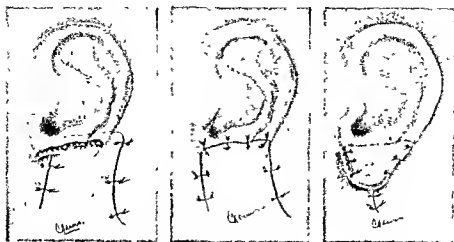


Fig. 99.—Lobule loss.

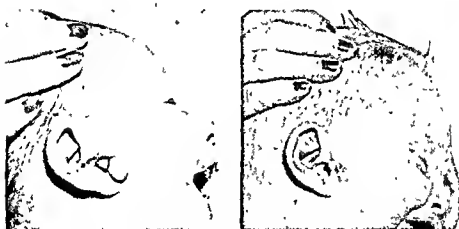


Fig. 100.—Anomaly of lobule and tragus.



Fig. 101.—Atresia of ear canal, Z plastic repair.



Fig. 102.—Atresia of ear canal. Skin graft repair.

flap to reproduce the helix. This procedure is utilized in constructing a total ear.

SUBTOTAL LOSS OF THE EAR (Pierce) (Fig 105)—Curved struts of preserved cartilage are planted through stab incisions which outline the helix of the ear and are allowed to organize. A tube of skin is fashioned along the upper margin of the clavicle. The mesial end of this is

draped about the newly constructed ear to form its helix.

Peer² implants diced, autogenous cartilage under the skin at the site of the ear construction. After its organization, he incises the outline of the ear, elevates the flap including the implanted cartilage, and grafts the mesial surface of this flap with thin split skin. The cartilage implanted

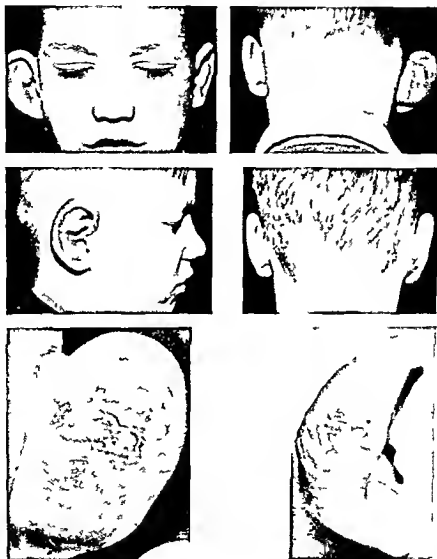


Fig. 103—Hemangioma (ear)

ultimately transplanted to the mastoid region to be utilized in constructing the helix.

After sufficient time has elapsed for the adhesion of the implanted struts to the covering skin, the helix is outlined by connecting the stab incisions, and the ear flap is elevated. Its mesial surface is covered with split skin. The inferior end of the tube of skin is freed and the tube

in the conchal region about the site of the ear canal is sufficiently thick to permit subsequent carving to produce gross contours.

CONGENITAL ABSENCE OF THE EAR EXCEPTING THE LOBULE (Peer²) (Fig 106)—The superior margin of the ear has been outlined by incision through the skin and the flap which will cover the external surface of the ear has been

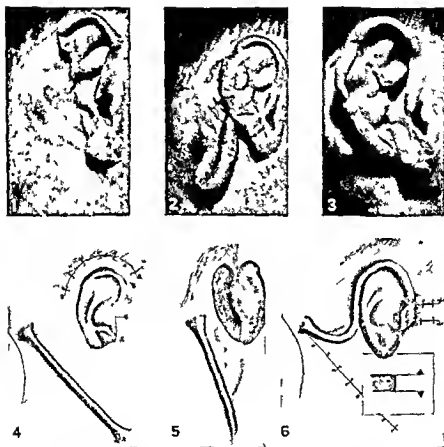


Fig 104 —Kirkham's ear operation (Courtesy of Ann Surg)

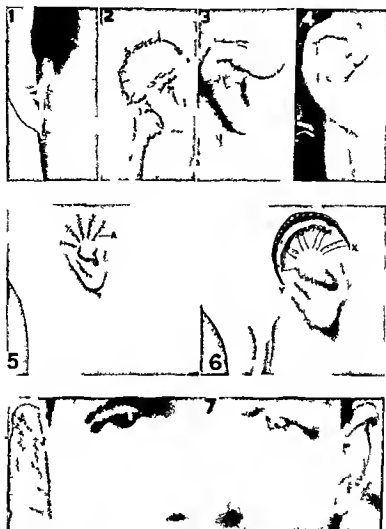


Fig 105—Pierce's operation on (ear) (Courtesy of Surg Gynec & Obst)



Fig 106—Peer's operation congenital absence of the ear excepting the lobule (Courtesy of Arch Otolaryng)

undermined down to the existing lobule. This pocket was packed with finely diced autogenous cartilage. A tube of skin which was fashioned above the clavicle has been transplanted as pictured. A pocket beneath the ear has been created by elevating the diced cartilage with its skin covering and this cavity has been lined with split skin (illustration on the left). In the right illustration, the ear has been completed by incision of the remainder of its border, the tube of skin has been draped about its margin to reproduce the helix and the implanted cartilage framework curved to simulate the external auditory canal and the adjacent structures.

FERRIS SMITH

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OPHTHALMOLOGY IN RELATION TO THE NOSE, THROAT, AND EAR

Anatomy of the Orbit—The orbit is formed of bony walls having the shape of a quadrilateral pyramid. The apex corresponds to the optic foramen. The base is directed forward and corresponds to the strong, thick projecting anterior margin. The nasal wall, the thinnest, is formed largely by the lacrimal bone of the os planum of the ethmoid, also by the frontal and sphenoidal bones. It presents in front a groove for the lacrimal sac. The inner walls of the orbit are almost parallel but the outer diverge considerably from each other from behind forward. The posterior portion of the orbit presents three openings leading into adjacent cavities: (1) the optic foramen, transmitting the optic nerve and the ophthalmic artery, (2) superior orbital fissures (sphenoidal) transmitting the ophthalmic vein, the nerves for the ocular muscles, and the first branch of the trigeminus, (3) the inferior orbital fissure (sphenomaxillary) transmitting the maxillary nerve and the intra-orbital artery. Besides communicating with the cavity of the skull by means of the openings at the apex, the orbit is surrounded by a number of other cavities. These are the nasal fossae and accessory

cavities: ethmoid and sphenoid sinuses, frontal sinus, and the maxillary sinus. These relations are important.

The contents of the orbit consist of the eyeball and optic nerve, the ocular muscles, the lacrimal gland, blood vessels, and nerves. The spaces between these are filled with fat and fasciae. The orbital fascia is extensive and presents numerous subdivisions. It serves as the periosteum to the wall of the orbit (peri-orbital). The portion closes in the opening of the orbit, forming an anterior wall and extending from the margin of the orbit to both tarsi and to the external and internal tarsal ligaments, thus constituting a *septum orbitale*. The prolongations of the orbital fascia surround the muscles and connect them with one another, the lids, and the margins of the orbit. In addition, a layer of fascia surrounds the globe from the cornea to the posterior part, separating the organ from the orbital fat. It is attached to the sclera by fine trabeculae. This investment is known as Tenon's capsule. Anteriorly, it merges into the subconjunctival connective tissue. Posteriorly, it disappears around the optic nerve and is pierced by all structures which are attached to the globe. In wide movements of the eyeball, both globe and its capsule move together as a whole upon the surrounding fat. Where the tendons of the ocular muscle pierce Tenon's capsule, the latter is reflected upon them and becomes continuous with their fascia.

The arteries of the orbit are derived from the ophthalmic artery. Veins empty into the ophthalmic veins which pass through the superior orbital fissure to the cavernous sinus. The nerves of the orbit are motor and sensory. The motor nerves, the third, fourth and sixth, supply the ocular muscles. The sensory nerves are the first and second branches of the trigeminus. The ciliary ganglion lies in the outer side of the optic nerve and receives its motor fibers from the third, the sensory fibers from the fifth, and the sympathetic filaments from the carotid plexus. It gives off the short ciliary nerves which enter the eye at its posterior part. The orbit contains no lymph vessels or lymphatic glands.

Congenital Anomalies of the Skull—According to Mann¹ the orbit must be considered as forming part of the whole skull and its deformities are linked with deformities of the skull rather than defects of the eye. As far as the eye is concerned, it is self-determining. Almost total failure of the orbits has been reported with an

eye of normal size and shape In anophthalmia (absence of the eye) the orbit is well formed though it may be slightly smaller than normal In microphthalmia (small eye) both the orbit and its contents, other than the eye itself, are usually normal

Oxycephalia (Tower Skull)—Tower skull is characterized by a high, narrow, pointed or dome shaped figuration of the skull, the anterior, posterior, and lateral measurements being diminished and the vertical increased The forehead is high, the eyes wide apart and protuberant, the orbits shallow and superciliary ridges deeply developed, the jaws often prognathous, and the palate arch high and narrow There is usually a certain amount of optic atrophy of the secondary type, and in some cases papille dema has been recorded Strabismus and nystagmus occur, but are secondary to the poor visual acuity In most cases the deformity is noticed at birth, in a few, it comes on gradually in from one to six years There is occasionally a history of convulsions in infancy and gradual failure of vision Headaches appear in childhood and usually cease at the age of eight when the growth of the brain slows down

Röntgen ray appearances are diagnostic Apart from the peculiar shape of the head, the most striking feature is the appearance of the bones of the skull These appear extremely thin and pitted all over with shallow depressions on their inner surfaces They are known as "digital impressions" The vessel markings are exaggerated, probably owing to the thinness of the skull Many of the sutures are prematurely closed, and, as a result of this, the middle fossa is often pushed down nearly to the level of the posterior fossa, and the anterior fossa is shortened and distorted In true oxycephalia, the transverse sutures (skull base, coronary, and lambdoid) are probably involved first The anterior fontanel may remain long and open, and may even gape, a cerebral hernia being produced here under pressure

In some cases the sagittal suture has been reported to close early, but it must be remembered that at least three types of skull deformities are caused by premature synostosis of sutures, and in many of the earlier reports these types are confused They are (1) oxycephalia (tower skull)—abnormally large, vertical measurements and short transverse anterior and posterior measurements—probably due to premature closure of transverse sutures and failure of

the bone growth in this region, (2) scaphocephaly (scaphoid head)—long, narrow dolichocephalic skull, the anteroposterior measurements being increased and the transverse diminished—probably due to early closure of the sagittal suture and sometimes confused with oxycephalia, (3) plagiocephaly (slanting head)—also confused with oxycephalia—due to various asymmetrical, abnormal closures

Hypertelorism—This is a rare condition in which the orbits retain their primitive lateral position, as a result of early ossification of the lesser wings of the sphenoid This fixes the orbits in the lateral (fetal) position As a secondary change the frontal and maxillary bones are pushed upwards, forwards, and outwards, and intranasal bone develops There is a low forehead with slight prominence of the frontal eminences Many associated defects are found including external strabismus, syndactylia, enlargement of the terminal phalanges of the thumbs, and undescended testicles

Abnormalities in the Number of Eyes—Cyclopia—Cyclopia is a condition in which a single median eye (or the two eyes in contact with each other or in various stages of fusion) is present The cyclops fetus may show the deformity developed to varying degrees and several forms of cyclopia have been described as separate conditions They all represent varying stages of suppression of the nose and the fusion of the two orbits The commonest are (1) two separate orbits and eyes very close together and a rudimentary nose between them with a single nostril (*cebocephalia*), (2) two separate orbits and eyes in contact with each other, the nose being represented by a proboscis like process above and between them (*arhinencephalia*), (3) a single median diamond shaped orbit containing two eyes side by side but not fused (there are usually four lids and the lacrimal passages are absent), (4) *multiple eye* in which, in certain monstrosities, more than two eyes may occur in apparently a single fetus The two commonest forms of multiple eye fall under the headings of "anterior dichotomy" and of "fused fetuses" Anterior dichotomy implies a bifurcation of the anterior end of the embryo at the early stage If it is complete, two separate heads, and therefore four eyes, will be attached to the same body If it is incomplete the eyes may be partially fused, four eyes being present, the two median ones lying close together, or even partially fused, so that, in appearance, three eyes

are produced. Posterior dichotomy is a bifurcation of the posterior end of the fetus. It is said sometimes to extend forwards as far as the basisphenoid region and to lead to the production of an accessory face here. In both anterior and posterior dichotomy a single ovum gives rise to more than two eyes.

Affections of the Orbit—According to May, affections of the orbit include exophthalmos and enophthalmos, periostitis, cellulitis, thrombosis of the cavernous sinus, tenonitis, tumors, injuries, and ocular manifestations of diseases of the ear, nose, and throat.

Exophthalmos is a protrusion of the eyeball from the orbit and is a common sign in affections of this region. It is caused by congenital malformations of the orbit, cellulitis, edema, inflammations, tumors and injuries of the orbit, enlargement of the eyeball, dilatation of the adjoining cavities, vascular anomalies, goiter, chronic nephritis, acromegaly, and in some cases is associated with paralysis of the ocular muscles. It is apt to produce conjunctival congestion and epiphora. When marked, it may cause interference with the mobility of the eyeball, imperfect closure of the lids, with a resulting keratitis from exposure, ectropion of the lower lid, diplopia, and, occasionally, interference with vision from optic neuritis.

Enophthalmos is a recession of the eyeball into the orbit. This is a rarer clinical condition, but is seen in markedly emaciated children as a result of a decrease in the orbital fat. Other causes are cicatricial contractions due to or following orbital injuries, cellulitis, operations, fractures of the walls of the orbit, and congenital defects.

Orbital periostitis is an inflammation of the orbital periosteum which may be either acute or chronic and limited to a portion of the margin of the orbit or spreading more deeply. Products of the inflammation often consist merely of thickening of the membrane, sometimes there is a deposit of bone or there may be formation of an abscess with or without subsequent necrosis of a part of the wall of the orbit. The most common variety is that affecting the margin of the orbit. This is characterized by tenderness on pressure on the orbital margin over the hard, immovable swellings. There is some edema of the lids and the conjunctiva. If there is pus, a subperiosteal abscess develops at the margin of the orbit which perforates the skin, leaving a fistula through which a probe

can follow the fistulous tract to the necrosed bone. The fistula may remain open for months or close and then recur. If the periostitis is situated more posteriorly, there will be more pain, deep seated in character and accompanied by tenderness on pressure on the globe. There is usually considerable edema of the lids and conjunctiva and exophthalmos. If the deep-seated process goes on to the formation of an abscess, it presents the signs of orbital cellulitis. Pus may find its way to the surface, but may infiltrate through the roof of the orbit and become a threat to life through extension into the cranial cavity with resulting meningitis or cerebral abscess. The disease is usually caused by injuries, tuberculosis, syphilis, or extension of affections of the nasal accessory sinuses. Of all causes, trauma is probably the most frequent exciting factor.

Orbital cellulitis is an inflammation of the cellular tissues of the orbit usually terminating in suppuration and may be called "orbital phlegmon" or "retrobulbar abscess." It runs more or less an acute course, generally accompanied by marked constitutional symptoms. There is a great swelling of the lids, chemosis of the conjunctiva, exophthalmos, impairment of the mobility of the eyeball, violent pain in the orbit increased by pressure upon the eyeball, and, usually, high fever. Vision may not be affected, but usually it is reduced and may be lost owing to the occurrence of an optic neuritis, followed by atrophy of the optic nerve. After these symptoms have lasted about a week, pus usually appears below the superorbital margin and a fistula develops. After evacuation of the pus, the symptoms subside leaving some permanent damage. The etiologic factor is frequently an extension of disease of the naso accessory sinuses, especially from the ethmoid, or from neighboring foci, such as orbital periostitis, infected teeth, injuries, foreign bodies, facial erysipelas, septicemias, and acute infectious diseases, especially influenza.

Tenonitis is an inflammation of Tenon's capsule and is manifested by moderate swelling and redness of the upper lids with diffuse chemosis and exophthalmos, and limitation or absence of ocular rotations. This usually follows injury or influenza. It is sometimes difficult to differentiate tenonitis from a typical orbital cellulitis, although it may follow severe forms of iridocyclitis and panophthalmitis.

Thrombosis of the cavernous sinus is almost

always infective and usually fatal, owing to the extension of the thrombus in the orbital veins. This may occur in conjunction with orbital abscess or neighborhood foci situated in the nose and throat, or secondary to erysipelas, caries of the petrous bone, empyema, or infective diseases. The signs and symptoms are similar to those of orbital abscess from which it can usually be differentiated by the existence of severe venous involvement and cerebral symptoms.

The ocular manifestations of diseases of the ear, nose, and throat comprise not only infections of the orbit and its contents due to extension, but may act as foci of infection. The accessory sinuses of the nose surround the orbit. They are separated by bony walls which are very thin in spots. They are lined by an extension of the nasal mucous membrane, and, as a result of such relationship, often become infected. When the sinus outlet is blocked, secretion will accumulate. This may lead to distention of the sinus, called the mucocele, with encroachment upon the orbit producing exophthalmos and other signs of orbital tumor. Such a sinusitis may run an acute or chronic course.

When the frontal sinus is involved there may be a bulging at the upper and inner angle of the orbit with tenderness on pressure over this area, and sometimes redness of the overlying skin, severe frontal headaches, and vertigo. There may be protrusion of the eyeball downward and outward, diplopia, edema of the lid, conjunctival and episcleral congestion, and lacrimation. Orbital periostitis and cellulitis may result.

When the ethmoid sinuses are involved, there may be a tumefaction at the upper and inner part of the orbit with swelling of the skin of the adjacent lids, displacement of the globe downward and outward with diplopia, marked pain, conjunctival and episcleral congestion, and lacrimation. The process may involve the orbit, producing periostitis or cellulitis. The infection may harbor the infective foci responsible for certain cases of uveitis and iritis.

Diseases of the sphenoid sinus are usually associated with ethmoiditis. The walls of this cavity and the optic nerve are continuous and this close relationship has been used to explain the occurrence of optic neuritis and retrobulbar neuritis. Medical literature is rather voluminous on this subject, but, in my own personal experi-

ence, sphenoiditis or posterior ethmoiditis rarely, if ever, produces retrobulbar neuritis or optic neuritis. When it does occur, it is by direct extension to the arachnoid through the embryonic form plate and produces a prechiasmal syndrome due to chronic basal arachnoiditis.

Diseases of the antrum do not often produce ocular complications, but they occur, especially in extension of infection of the teeth into the antrum, producing an orbital periostitis or cellulitis. Tumors of the antrum invade the floor of the orbit by direct extension and produce secondary ocular complications.

Tumors of the orbit are of infrequent occurrence and they may arise from the walls or contents of the orbit or spring from the neighboring cavity. The symptoms depend upon the size, position, and the nature of the tumor. Exophthalmos is usually present and the direction of the protrusion and the impairment of the motion of the eyeball are usually dependent upon the situation of the tumor. Pressure upon the optic nerve may cause papilledema and later optic atrophy.

Benign tumors of the orbit include dermoid cyst, neurofibroma, aneurism, angioma, meningocele, mucocele or pyocele from the neighborhood cavities, and osteoma. Malignant tumors are sarcoma and carcinoma. Intraocular sarcomas are usually primary tumors and metastasize elsewhere. Intraocular carcinomas are always metastatic and many have their primary lesion in the bronchi. Carcinoma of the eyelids proceeds by direct extension and many times involves the bony walls of the orbit and invades the nasal accessory sinuses.

Injuries of the orbit include contusions, penetrating wounds, foreign bodies, and fractures of the orbital wall. The prominent sign is a hemorrhage in the orbit, causing exophthalmos and sometimes ecchymosis of the lids and conjunctiva. A stab or blow with a blunt object may rupture the globe or occasionally dislocate the eyeball in front of the lid. This displacement is sometimes produced through gouging with the thumb. Fractures may involve the anterior margin and may be detected by an unevenness on palpation. A fracture of the inner wall may cause emphysema. In fracture of the apex involving the optic canal, optic atrophy may develop as a sequela. Fracture of the lower bony margin of the orbit usually produces a vertical diplopia because of involvement of the inferior oblique muscle.

Intracranial Affections of the Visual Pathways Associated with Diseases of the Ear, Nose, and Throat.—Prechiasma syndrome is characterized by retrobulbar neuritis which may be acute or chronic in its course. Retrobulbar neuritis, in turn, is characterized by diminution of central visual acuity, normal fundi, and visual fields showing some form of a central scotoma. The visual field defect is always on the side of the lesion. The commonest type of lesion producing such a syndrome associated with nasal pathologic change is *chronic basal arachnoiditis*. Tumors of the olfactory groove produce the same type of syndrome and have an associated diminution of sense of smell on the side of the lesion. A chiasma syndrome is associated with an extension of nasopharyngeal disease process through the cranial pharyngeal duct affecting the optic chiasm in the region of the pituitary gland. The initial ocular changes are paralysis or paresis of one or both sixth nerves producing an external rectus palsy associated with diplopia. General signs of pituitary dysfunction may also accompany this in the form of increased weight, absence of menses in women, and impotency in men. There may be a vaulting of the roof of the nasal pharynx which is visible by examination. These occur because the anterior portion of the pituitary gland has its anlage in the buccal mucosa and congenital rest cells remain along the craniopharyngeal duct.

Petrositis produces ocular complications in the form of severe one sided pains, paroxysmal in type, because of involvement of the fifth nerve and in rare instances there is a sixth-nerve paralysis on the same side.

The *cerebellar pontine angle syndrome* is characterized by tinnitus and deafness on the side

of the lesion and is associated with a diminution of the corneal reflex on the same side. This occurs as frequently as deafness in about 98 per cent of the cases. Later a sixth and seventh nerve paralysis may develop and in only 50 per cent of the cases is there a choking of the discs. Intrapontine lesions never produce deafness. The tympanic membrane on the side of the deafness in a cerebellar pontine angle lesion also shows a diminution of sensitivity, comparable to the corneal diminution of sensitivity. It is the result of pressure on the long, posterior descending sensory root of the fifth nerve and is a clinical manifestation which aids the otologists in differentiating cochlear nerve deafness from deafness due to a lesion situated in the cerebellar pontine angle.

Nystagmus is not an infrequent occurrence in diseases of the labyrinth and cerebellum but has no localizing value from an ophthalmologic standpoint and in itself is not significant.

A homolateral sixth nerve paralysis sometimes accompanies an acute otitis media (Gradenigo's syndrome) and mastoiditis. Choked discs may also be associated with acute otitis media and mastoiditis.

Intracranial complications of mastoiditis may affect the visual pathways. These include meningitis, temporal lobe and cerebellar abscesses.

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PART II. MOUTH, FAUCES, AND PHARYNX

APPLIED ANATOMY OF THE PHARYNX

Definition.—The pharynx is the common passageway for air and food extending from the nasal and oral cavities to the esophagus and

the torus tubarius, consisting of cartilage covered by mucous membrane. Above and behind the torus is a recess called the fossa of Rosenmüller. On the roof of the nasopharynx, in childhood and sometimes persisting into adult life, is the pharyngeal tonsil or adenoid.

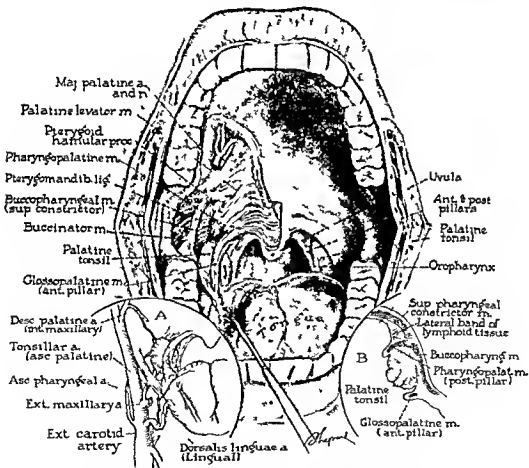


Fig 107—Surgical anatomy of the pharynx, palate and tonsils

larynx. The pharynx is divided into the nasopharynx, oropharynx, and hypopharynx.

The Divisions of the Pharynx.—*Nasopharynx*—The nasopharynx lies above the level of the soft palate, and has five openings: the two posterior nares, the two eustachian tube orifices, and the opening downward into the oropharynx. Each eustachian tube orifice is surrounded above and behind by a prominent bulge called

Oropharynx—The oropharynx lies between the soft palate and the upper edge of the epiglottis, and has three openings: above to the nasopharynx, below to the hypopharynx, and anteriorly to the oral cavity. The fauces separate the oropharynx from the oral cavity, and consist of the soft palate and uvula above and two folds extending from the soft palate to the tongue on either side, called the anterior and

posterior pillars of the fauces. The faucial tonsil lies between the anterior and posterior pillars.

Hypopharynx.—The hypopharynx lies below the upper edge of the epiglottis and has three openings: above to the oropharynx, below, anteriorly, to the larynx, and posteriorly to the esophagus.

Mucous Membrane.—The pharynx is covered by stratified squamous epithelium except for the roof and lateral walls of the nasopharynx which are covered with ciliated columnar epithelium continuous with that of the nasal cavities. Mucous glands in the subepithelial con-

nor pillar draw the palate downward and with the help of the base of the tongue, close off the oropharynx from the oral cavity. The muscles of the palate are also innervated by the vagus nerve.

Lymphoid Tissue and Lymphatics of Pharynx

—Superficial accumulations of lymphoid tissue characterize the pharyngeal mucosa and consist of (1) the faucial tonsils, or palatine tonsils, (2) the adenoid, or pharyngeal tonsil, (3) the lingual tonsil, (4) the lateral pharyngeal bands, (5) solitary lymphoid nodules scattered over the posterior pharyngeal wall. Together

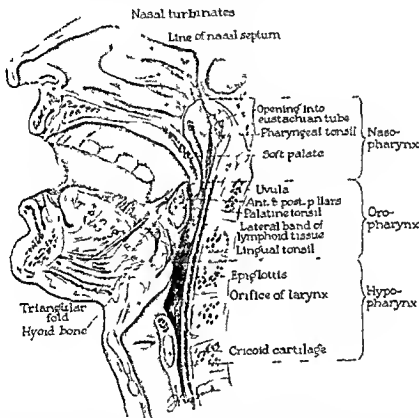


Fig. 108.—Sagittal section showing outer wall of the right nasal cavity, nasopharynx, palate, pharynx, and larynx.

nective tissue are especially numerous in the upper part of the pharynx. The sensory nerve to the pharynx is the glossopharyngeal.

Muscles.—The walls of the pharynx are formed by the superior, middle, and inferior constrictor muscles of the pharynx, innervated by the vagus nerve, for the purpose of deglutition. The soft palate contains the levator palati and tensor palati muscles to raise the palate and close off the nasopharynx from the oropharynx and to open the eustachian orifices. The palatoglossus muscle in the anterior pillar and the palatopharyngeus muscle in the poste-

rior pillar draw the palate downward and with the help of the base of the tongue, close off the oropharynx from the oral cavity. The muscles of the palate are also innervated by the vagus nerve. Lymphoid tissue constitutes Waldeyer's ring. Its function appears to be to localize infections that enter the body through the oral and nasal orifices, and perhaps to aid in the acquisition of immunity to organisms entering the mouth and nose, for hypertrophy of this lymphoid tissue occurs normally in childhood during the years that immunity is being acquired to many organisms and infectious diseases. Beginning about the time of puberty the pharyngeal lymphoid tissue begins to atrophy, this atrophy progressing gradually through adult life except in the presence of chronic in-

fection when hypertrophy usually occurs and persists

The Adenoid—The pharyngeal tonsil, or adenoid, lies on the roof and upper posterior wall of the nasopharynx. It consists of lymphoid tissue with five or more fissures running antero-posteriorly and many tubular crypts extending in from the fissures and ending in mucous glands, the lymphoid follicles with their germinal centers being arranged along the sides of these fissures and crypts. The adenoid, its fissures and crypts, are covered with ciliated columnar epithelium which keeps the fissures swept clean of debris. There is no well-defined fibrous tissue capsule separating the adenoid from the superior constrictor muscle, so that surgical enucleation of the adenoid with complete removal of all lymphoid tissue is not possible, and regrowth of adenoid tissue occurs frequently following adenoidectomy. Adenoid tissue may extend into the fossae of Rosenmüller and over the torus tubarius.

The Tonsil—The faucial tonsil lies between the anterior and posterior pillars of the fauces. It consists of lymphoid tissue with 8 to 20 tubular crypts extending from the surface to the depth, these crypts frequently branching in the depth of the tonsil. Lymphoid follicles with their germinal centers are arranged in a row along the sides of the crypts. The surface of the tonsil and the crypts are clothed with stratified squamous epithelium. Desquamated epithelial cells, lymphocytes and a few leukocytes normally collect in the crypts as a caseous-appearing plug of debris which may be expressed by pressure on the anterior pillar.

The faucial tonsil is surrounded by a layer of connective tissue forming a capsule separating the tonsil from the constrictor muscles of the pharynx. This capsule is very clearly defined over the superior pole of the tonsil, and is thinner and less evident over the lower pole. A triangular fold of mucous membrane, the plica triangularis, stretches across the lower anterior surface of the tonsil from the anterior pillar to the base of the tonsil fossa. The blood supply of the tonsil comes from the lingual artery which sends a branch to the lower pole, from the external maxillary artery which sends a tonsillar branch to midway between the upper and lower pole with smaller branches from the ascending pharyngeal artery, from the ascending palatine artery, from the descending palatine artery, and from a few minor branches of

the small meningeal artery. All of these vessels are from the external carotid artery. The tonsil capsule makes possible complete enucleation of all tonsillar tissue by splitting the capsule. After such a successful operation the tonsil cannot reform, although nodules of superficial lymphoid tissue such as occur on the posterior pharyngeal wall may later appear in the tonsil fossae.

Lingual Tonsil, Lateral Pharyngeal Bands, and Scattered Lymphoid Nodules—On the base of the tongue the lingual tonsil consists of lymphoid tissue containing shallow tubular non-branching crypts lined by stratified squamous epithelium. In the lateral wall of the pharynx, just behind the posterior pillar, there is often an accumulation of superficial lymphoid nodules, called the lateral pharyngeal bands. On the posterior wall of the pharynx are scattered superficial lymphoid nodules. Both the lateral bands and the scattered nodules are more developed in the upper part of the oropharynx and less developed inferiorly.

Lymphatics—The lymphatics of the pharynx are abundant and drain into the anterior cervical lymph nodes, anterior to the sternocleidomastoid muscle, except those from the nasopharynx and posterior oropharynx some of which drain into the posterior cervical lymph nodes posterior to the sternocleidomastoid muscle. In infancy there are several lymph nodes in the connective tissue between the upper cervical vertebrae and the superior constrictor muscle of the pharynx. Lymphatics from the pharyngeal mucosa drain into these retropharyngeal lymph nodes. These nodes disappear soon after infancy.

GEORGE E. SHAMBAUGH, JR.

DISEASES OF THE MOUTH AND TONGUE

Congenital Malformations.—Before taking up diseases of the oral cavity, it would be in order to consider congenital malformations. Of these there are many, some of which are rarely seen, while others are quite common—or at least are more often observed by the medical practitioner. Therefore, these are of greater interest

from the standpoint of human happiness and of possible surgical correction

Hare Lip and Cleft Palate — *Harelip* (cleft lip, *lagocheilus*, fissure of the lip) is probably the most frequent congenital cleft and is often

cleft In double cleft, for example, we most often have protrusion of the premaxillary bone and double cleft palate Many variations have been described and illustrated in books devoted to the subject



Fig 109

Fig 109 — Cleft lip with central portion and nondevelopment of nasal bones



Fig 110

Fig 110 — Nondevelopment of premaxilla and midportion of lip

found with fissures of the alveolar process or clefts of the palate They are not always associated however as one may be found without the other Cleft lip may consist of a small notch or groove or may be so extensive as to include the whole lip or even extend up to and include

Cleft palate may be classed as single, when the cleft extends backwards in midline and the nasal septum is attached to one side of the cleft, and double when the nasal septum is not attached to either side but is a separate structure in the middle with a cleft on each side The



Fig 111

Fig 111 — Unusual cleft But the isthmus between the posterior and anterior cleft is shown Rare



Fig 112

Fig 112 — Inoperable cleft palate — rare



Fig 113

Fig 113 — Section of tongue engrafted in cleft palate giving good speech

the nose and eyelids The fissure may be upon one side only (single) or on both sides (double), or, rarely, in the midline only Cleft of the lower lip is always midline and is also rare

The extent of the deformity depends upon whether the alveolar process or palate is also

latter condition is most often associated with cleft lip Cleft palate may be of various degrees from cleft of the soft palate only, or simply a *bifid uvula* to the clefts of the hard and soft palate just mentioned

The cause of harelip and cleft palate is un-

known although heredity probably plays an important part. In over 2000 cases we have found that in only 40 per cent could any previous family history of deformity be elicited. In many of these the mendelian law seems to be followed. Malnutrition, because of pernicious vomiting in the mother during pregnancy, has apparently been the cause in some cases. This may, however, be questioned. Syphilis is sometimes given as a cause, but my associates and I have made a series of serological tests on the child, mother, and father in our cases. These have all been negative. Berry and Legg state that it often appears at the beginning or end of large families. No one of these causes can be

The time and age when the operation is best depends largely on the condition of the patient. A child may be robust enough to be operated upon in the first two or three weeks of life or may require the care of a pediatrician to build up his health. The food factor is very important and in many cases, much better results are obtained by waiting. We have found, however, that if the cleft is wide the baby may swallow so much air with the feeding that a large portion is regurgitated, in some of these cases closure of the lip may cure the air swallowing tendency. In other cases we have used adhesive strips to narrow the cleft. This will also prevent some of the air swallowing. Much better



Fig 114—A Single cleft lip before operation B after operation and immediately after removal of sutures

made to apply to all cases. Multiple cases in one family are frequently seen, but these children are not often consecutive births. Probably a defect in the sperm or ovum cells at the time of fertilization, is the key, but this cannot be proven with our present knowledge. A record of these cases can best be kept for filing by using the chart of Dr Harry Ritchie which I have found invaluable.

SURGICAL TREATMENT—In no case of plastic surgery is operation more difficult and in none does so much depend on a good result as to both appearance and physiologic action. It requires sound judgment on the part of the surgeon to decide when to operate and in what cases not to operate.

results will be obtained both to life and to cosmetic appearance if the patient is in good physical health.

These little children may have other deformities and are not always good anesthetic risks. We have found that a large percentage have thymus enlargement and will frequently die if given an anesthetic. We, therefore, have all patients showing an enlargement of the thymus treated with deep roentgen therapy until the thymus returns to normal size when operation can be performed safely.

Operations on the palate may be performed when the child is between six and eighteen months of age. My preference is usually between ten and eighteen months or before the

child learns to talk. Dorrance prefers to wait until the child is five years of age or older.

The operations of Brophy and Lane have not been successful in my hands, many others have also abandoned these procedures.

The *cleft lip operation* depends on the extent of the cleft, many times an incomplete cleft may show lack of muscle fibers in the upper part which will allow the nostril of the affected side to sag. In this case, it is necessary to cut through this isthmus of tissue and make the cleft complete (Owen Smith operation). The incision is first made along the junction of the skin and vermilion border through about two thirds of the thickness of the lip. This flap is made upon both sides, but the extent of the incision is varied so that the distal side is longer. These flaps are then folded inward and sutured on the

removed or the septum incised and the ends of the fragments beveled to slide over each other and sutured with catgut. It may also be necessary to use flaps of the mucoperiosteum, as in Brophy's technic, in order to bring bone surfaces together or healing will not take place. Simply forcing the premaxillary bone into a normal position will not make it fuse. Even with this procedure, it may be necessary to use a suture of silver wire to hold the bone in place for about six weeks.

Cleft palate operations require a special technic. The first step, if the cleft is complete and involves the alveolar process, is to dissect the mucoperiosteum from the bone. Beginning at the edge of the hard palate immediately anterior to the posterior border at the junction with the soft palate, it is dissected outward to the



Fig 115—Double incomplete cleft of lip, A, before operation, B, after operation, C, aged two years

oral surface with chromic gut, preferably 20 day. With atraumatic needle, the skin edges are then sutured with silk, a slightly heavier suture being used for the floor of the nostril, with finer silk for the remainder, unless the cleft is wide, when heavier silk may be used. In very wide single clefts a silver suture with shot or lead plates may be used through the lower border of the alar cartilage and through the nasal septum. This method gives extra support and has a tendency to narrow the alveolar cleft. In cases of narrower clefts an arched support may be used over both alar cartilages and kept in place with moleskin adhesive.

In cases of *double cleft* of the lip the technic is similar, except when the premaxillary bone is protruding, in which case a section of the lower part of the nasal septum must either be

alveolar process laterally, and forward to and including the anterior alveolar process cleft. This is repeated for the opposite side of cleft. There is now a pocket on each side below the palate process of the maxilla which is still attached to the soft palate posterior. In order to allow the tissue to be depressed, the soft tissue at the posterior border of the hard palate (the horizontal process of palate bone) is incised on the nasal aspect and the incision carried laterally to the tuberosity. If relaxation is sufficient, this will allow the edges of the mucosa of the soft palate and the periosteum of hard palate to be contacted by means of thumb forceps. If relaxation is not sufficient, a circular incision around the tuberosity on each side will be necessary.

The soft palate is now split from the tip of

the uvula to the anterior junction with the hard palate. The oral and nasal flaps thus formed are sutured beginning at the tip of the uvula, using chromic gut for the nasal flap and silk for the oral flap. The silk suture is now continued forward bringing the mucoperiosteal flaps of the hard palate in contact and everting the edges, this is best accomplished with interrupted mattress suture.

After-care—The *treatment of wound surfaces* is best accomplished by careful irrigation of oral surfaces with warm saline solution. It is well to use drops of the same in each nostril. The best results are obtained by giving this treatment after each feeding.

The greatest care in *feeding and nursing* after the operation is as important as the operation itself. Liquid food, but not milk, should be

also be conducive to infection, constant prone position being conducive to ear infection. Therefore, frequent changes in position or holding by the nurse are advisable.

In older children the care of the teeth both before and after the operation is of the greatest importance. While restraint of the hands is not so necessary, we have found that the tongue action on sutures may be detrimental and may be controlled by protection of the suture line by the prosthetic appliances suggested by Mackenty. Restrictive action of the tongue can also be accomplished by a suture for the first twenty four hours or longer. These patients should be examined at regular intervals by the pediatrician and surgeon and should be protected as much as possible from infectious diseases.



Fig. 116—Double cleft lip before and after operation

given for the first three days. * If milk must be used, careful cleansing must be meticulously carried out. Sutures should be removed in seventy two hours although part of them may be left for forty-eight hours longer. If 20 day chromic gut is used for both nasal and oral flaps, it may be allowed to remain until absorbed. This we have found much better when the operation is on a younger child.

The bands of younger children must be restrained or splinted so that they cannot reach their mouths since all previous care in feeding and the operation itself can be nullified in one second. The position of the child in bed may

The palate cases should be watched for tonsil infection. If these organs are to be removed it must be an especially careful operation to avoid injury to the pillars since a good result on a palate may be ruined by a poor operation on the tonsils and adenoids. *Speech training* is very important and should be begun at the earliest possible time after operation.

Other Congenital Oral Malformations—*Macrostomia*, or large mouth, is another deformity of the mouth. This is usually an abnormal extension of one or both angles. *Microstomia*, an abnormally small mouth, is rare, but does occur. *Double mouth and tongue*, as in the case reported by Dr. Hugh Beatty, is very rare.

Associated Deformities—At times, congenital deformities of the oral structures are accompanied by other congenital malformations such

* In infants and young children we find that both palate and lip heal better if no food or water is given by mouth. These children are given 10 per cent glucose continuously by vein for the first five days.

as spina bifida club foot deformed hands and feet, absence of fingers and toes, as well as webbing of either hands or feet. A few of the patients I have seen have been hydrocephalic

the premaxillary and of the nasal bones may complicate these clefts and I have also seen two other patients in whom only one half of premaxilla was present



Fig 117



Fig 118



Fig 119

Fig 117—Macrostomia with underdevelopment of ear

Fig 118—Double mouth and two tongues (Case of Dr Hugh G Beaty)

Fig 119—Left half of premaxillary developed with the right half absent. Underdevelopment of eyes. There is microphthalmia also in this case

two were affected with mongolism and two with microphthalmia. In one of the latter only one half of the premaxillary bone was

Mouth and Tongue Injuries—Injuries to the lip and oral mucosa may take place by mechanical, thermal or chemical means. Mechanical wounds



A



B

Fig 120—A Mandibular recess in case of cleft lip and palate. This patient had multiple deformities including deformity of feet and toes. B Ten years later

present and only one half of the central portion of the lip. Mandibular recess is a deformity of the lower lip which is also found associated with clefts of the lip or palate. The absence of

may be incised, lacerated, or contused, the latter most frequently involve the bony framework of the mouth or face and may be simple or compound. In clean incised wounds, it may

only be necessary to bring soft tissue in coaptation with fine suture whether it be on skin or mucosa but if the wounds are lacerated it may be necessary to cut away any tissue which is in danger of losing its blood supply or becoming infected. It is important to remove all clots, cleanse the wound with an antiseptic solution and replace bone or cartilage in as nearly normal position as possible. In most cases immediate suture is best but if the patient is severely shocked it may be better to wait until the patient has been treated for the more serious conditions since manipulations of parts either with or without an anesthetic may be detrimental to the life of the patient while plastic work may be postponed without endangering the

Fracture of the maxilla or mandible requires special technics and may be very simple or may require much ingenuity in adjusting and in preparing special apparatus. Wiring of teeth in occlusion is the simplest procedure if both jaws are not fractured but the more complicated cases may require the help of the dentist together with or preceding plastic repair.

Diseases of the Mouth and Tongue—Stomatitis—Frequently various forms of oral inflammation are extensions of inflammatory lesions of the skin while at other times they are manifestations of systemic disease originating in some other organ. Herpetic lesions of the lips may extend to the mucous membrane of the mouth or vice versa. Simple catarrhal stomatitis



Fig. 121—Avitaminosis. A Before treatment. B after intravenous thiamine.

result. Wounds should be cleansed and hemorrhage stopped. At present we believe it best to use locally one of the sulfa drugs or penicillin.

Infection of the lip or oral mucosa may occur due to a break of some sort in the continuity of the membrane caused by an injury its severity depending upon the virulence of the infective organism. While it may be impossible to demonstrate a break in the continuity of the membrane in every case of infection it is probably always present and many times can be shown conclusively. This is especially true of infections in the mouth where organisms frequently gain entrance around a rough tooth or artificial tooth replacement because the gum tissue is irritated. In affections of the bony framework, a history of trauma is always present

may be a forerunner of more severe forms which manifest themselves by ulceration.

APHTHOUS STOMATITIS—This condition is found most frequently in children and young adults and is characterized by ulceration on the mucosa of the lips and tongue. As a rule it is associated with gastric disturbances. The ulcers are shallow but painful and may require palliative treatment although they will heal readily when other parts of the alimentary canal return to normal.

PARASITIC STOMATITIS (THRUSH)—This is found in children and is due to *Oidium albicans*. Treatment with gentian violet in addition to hygienic measures will usually clear it up in a few days.

VULCANITE STOMATITIS—Many variations of stomatitis are due to traumatic injuries of the

cheeks or tongue in mastication. *Vulcanite stomatitis* is traumatic in origin, partially due to injury from ill-fitting plates, and partially to chemical irritation from some chemical in the composition of the denture (almost always rubber). *Treatment* consists of removing the denture, limiting its use, or fitting the patient with new dentures of different material.

STREPTOCOCCUS STOMATITIS—This condition consists of a marked redness of the mucosa and at times ulceration. *Streptococcus* is the predominant organism and may be associated

grown under anaerobic conditions. *Diagnosis* is usually easily made from the clinical symptoms, but it is always best to make a smear. The organisms may be found, however, in normal mouths and in other lesions. *Treatment* consists of local cleansing with oxydizing agents, hydrogen peroxide and sodium perborate, and applications of copper sulfate in a 15 per cent aqueous solution. The patient is given a 25 per cent glycerin solution to apply after meals, cleansing food particles from the cavity first. Some patients respond slowly, for these, neosal-



Fig 122 —Noma A before surgery B, after surgery C twenty years later

with pharyngitis and tonsillitis. *Treatment* consists of hygienic measures with hot mouth-washes and gargles.

VINCENT'S STOMATITIS—*Vincent's stomatitis* is also called *Vincent's infection*, *Vincent's angina*, or *ulceromembranous gingivitis*. It may affect only the gingival margins or may spread to the cheeks, tongue, and pharynx. It usually begins in gum tissue near a poorly fitting crown, filling, or a tooth cavity. The cause is *Vincent's spirillum* associated with the fusiform bacillus. It is easily stained but must be

varsan or bismuth sodium tartrate may be given intramuscularly.

GANGRENOUS STOMATITIS (NOMA)—*Gangrenous stomatitis*, while rare, may follow acute infectious diseases in poorly nourished children. It is characterized by the death of a mass of tissue, usually of the cheek, which suddenly turns blue and then black and involves the whole thickness of the cheek or lip. *Treatment* is surgical if the condition has progressed to the necrotic stage. Possibly, with our new chemical sulfa compounds or penicillin, the infection

may be delayed or cured before this stage is reached. The use of vitamins may be of value as many of these patients show symptoms of avitaminosis.

While osteomyelitis may follow any surgical procedure, it occurs most frequently after the removal of an infected third molar tooth. This is probably due to the stripping of the periost-

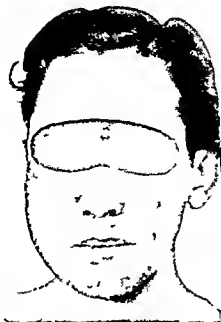


Fig 123—Osteomyelitis of mandible right body and ramus

Osteomyelitis—This is an inflammatory reaction affecting bony structures. It may affect either the maxilla or mandible, more frequently the latter. The infection most often originates

in an apical or pyorrhetic abscess of a tooth, and follows either accidental or surgical trauma. Drainage following surgical procedures must be carefully taken care of since poor drainage may be responsible for the infection of normal bone

teum and interference with the nutrition of the bony tissue, as well as to infection. The extension of the infection to the tissues of the floor of the mouth may involve the cellular structure



Fig 124—Osteomyelitis of mandible from abscessed tooth. Removed complete except for one condyle

in an apical or pyorrhetic abscess of a tooth, and follows either accidental or surgical trauma. Drainage following surgical procedures must be carefully taken care of since poor drainage may be responsible for the infection of normal bone

and the glands causing symptoms of Ludwig's angina, although one side of the mouth only may be involved.

Osteomyelitis of the maxilla may be caused by an abscessed tooth, or may also be caused

by maxillary sinus infection of nasal origin. In this case other sinuses may be involved and progressive osteomyelitis of the other bones of the face and skull develop. Two cases have come under my care in which the infection originated in the maxilla and fulminating osteomyelitis affected the ethmoid nasal bones and frontal sinuses with epidural abscesses.

The treatment consists in getting good surgical drainage either through the original wound or a new opening. Cultures should be made of the organisms present. General supportive treatment possibly including the giving of transfusions and a healthy regimen with proper elimination will aid recovery. After the organism or organisms have been isolated, chemical treatment should be instituted with one of the

The abscess may be located at the base of the tongue so the lingual tonsil or in midline; if in the latter position it is probably at the lingual

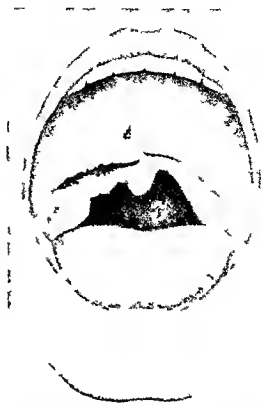


Fig. 126—Gummas of palate. Three perforations which later coalesced leaving one large perforation (Case of Dr. Daniel Newman)



Fig. 125—Abscess of tongue

sulfa compounds or, if possible, with penicillin. The use of penicillin has given excellent results in most cases, but it must not be expected to remove necrotic bone which requires surgery. The application of any of these remedies and whether a general or local effect is more suitable in a given case are best left to the judgment of the surgeon.

Abscess of the Palate and Tongue—An abscess of the palate may be produced by injury which introduces infection from tonsillar or tooth infection. In case it is from a tooth it may be from a pyorrhea pocket or the palatal root of a molar tooth.

Abscess of the tongue may be found in the midportion of the tongue, on the dorsum or on the border, and in any case is due to injury

end of a patent thyroglossal duct so that when it is opened a probe may be passed downward to the isthmus of the thyroid gland or into the anterior part of the neck where a swelling in

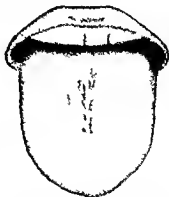


Fig. 127—Hairy or black tongue.

the midline may be found. Infection of the tongue which produces an abscess may be due to an injury by a foreign body, to simply biting the tongue, or frequently to irritation caused by

some substance such as a straw or toothpick being held in the mouth

Chancre of the tongue is not infrequently seen, it is usually on the edge or tip while a gumma is more often found in a middorsal position. A tuberculous abscess may appear on the tongue either on the edge, tip or dorsum,



Fig. 128—Tuberculosis of tip of tongue.

but more often it has ruptured and only the consequent ulceration is seen on examination

Glandular Cysts and Calculi—Swellings under the tongue may frequently be caused by cysts of the submaxillary or sublingual glands. Calculi in one of these glands or their ducts may simulate a tumor or cyst by interfering with the flow of saliva. The parotid gland may also be affected with calculi either in the gland



Fig. 129—Chancre of tongue

or in the duct, which may be confused with a mixed tumor or cyst. Roentgen ray may be used in making a diagnosis, but some calculi do not cast a shadow dense enough to show on the film. In the case of calculus, however, the swelling in any one of the salivary glands is usually found at mealtime or when the patient visualizes some especially appetizing food.

If the calculus is in the duct, slightly enlarging the orifice with a probe may allow it to be discharged, but if the calculus is in the gland, or if it is a case of multiple calculi, it may be necessary to remove the gland.

Ankylosis—Ankylosis of the temporomandibular joint may be due to an abscess in the



A



B

Fig. 130—A Ankylosis of right temporomandibular joint following abscess of upper third molar

middle ear, to furunculosis of the external auditory canal, to an abscessed upper or lower third molar tooth, to infection with abscess formation in the pharyngeal or parapharyngeal space or to inflammation about the joint as in actinomycosis, also fibrous tissue in cheek which may not involve joint. Ankylosis may be fibrous (temporary) or bony (permanent).

Treatment consists of breaking up scar tissue. If large scars are removed plastic replacement may be necessary. In the bony type of ankylosis when resection of the condyle of the mandible

early in life, or ankylosis of the temporomandibular joint. In the *treatment* of this condition, radiation therapy has been used with varying results and the sulfa drugs have been credited with some cures. The best results, however, I have obtained by the administration of iodine in the form of Lugol's solution, increasing the doses even to the extent of 1 ounce daily. Local treatment with copper sulfate crystals as used by veterinary surgeons or cauterizing with endothermic current or actual cautery may be employed.

Benign Tumors—The most frequently found tumors of the benign group are the fibromas, these may be either hard or soft depending on whether fibrous tissue or cellular elements predominate. Other benign tumors, less often found in the mouth, are osteomas, angiomas, lipomas, papillomas, giant-celled tumors, and neurofibromas. Any tumor on the gum may be called an epulis, but this is not a descriptive term and should be eliminated.

The location of tumors varies with the type. Fibromas and osteomas are found on the alveolar process while angiomas, lipomas, and papillomas are most frequently found on lips, tongue, and palate. There can be no hard and fast rule, however, since extension of the growth may take place to contiguous tissues although not to the same degree as in malignant growths.



Fig 131—Actinomycosis

and formation of a new joint are done, it may be necessary to implant muscle between the bone and the glenoid fossa to prevent regeneration of new bone.



A



B

Fig 132—Actinomycosis A, Before treatment, B after treatment

Actinomycosis—Actinomycosis as it affects the jaws will, as a rule, cause multiple abscesses which in healing will leave scar tissue which may cause deformities, if the disease occurs

It is probably unnecessary to mention that angiomas are frequently found at birth and may extend from their original border in almost any direction. Because of this extension they may



Fig 133 —Angioma of gum



Fig 134 —Tumor of gum



Fig 135 —Tumor of gum



Fig 136 —Cyst of maxilla

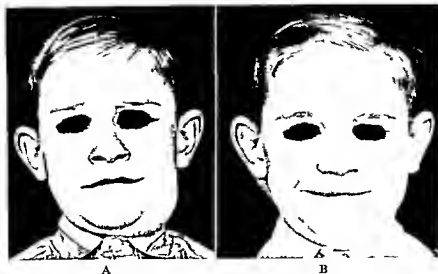


Fig 137 —Cyst of floor of mouth A, Before operation, B after operation

interfere with function or may be disfiguring if they are on the lip or face

Roentgen ray and radium may be used to restrict the growth. They may even cause the

exposed or because flaps can easily be made of contiguous membrane or skin

Growths on the palate may be any variety of tumor. An osteomatous growth in the midline



Fig 138—Thyroglossal cyst showing on anterior surface of neck A Before operation B after operation

disappearance of small lesions and in the case of larger growths they are a preface to any operative procedure. In larger growths constriction of the surrounding vessels may help by the

known as torus palatinus may interfere with the fitting of dentures

Papillomas may be found in various locations in the mouth more frequently on the tongue,



Fig 139—A Cyst of mandible B The patient four years later showing complete development after surgery

production of scar tissue to limit the borders so that complete resection can be carried out with out great deformity. This will, if successful make for easier plastic repair, immediate or delayed. In the case of small tumors no plastic repair is necessary because of the small area

soft palate, tonsil and lip, less frequently on the gums and the floor of the mouth

Malignant Growths—Carcinomas and sarcomas are found all too frequently in the mouth

SARCOMA—A sarcoma is a connective tissue tumor. The type of sarcoma depends on the



Fig 133 —Angioma of gum



Fig 134 —Tumor of gum



Fig 135 —Tumor of gum



F g 136 —Cyst of maxilla

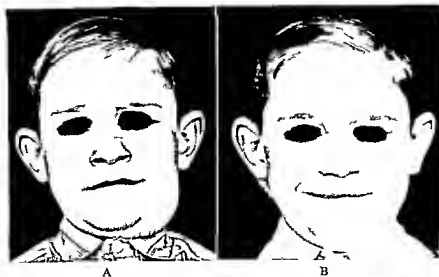


Fig 137 —Cyst of floor of mouth A, Before operation B after operation

the pathologist report confirms the malignancy. No operation should be attempted unless the surgeon is prepared for complete removal of the maxilla or, at least, one half of mandible. In cases where the growth has been present for some time or is increasing rapidly, metastasis may already have occurred and local operation is of no value.

certain. A close examination, however, will reveal to the experienced physician the possibility of injury to the cheek, tongue or lips which may have taken place during mastication or by other ordinary means.

To tobacco, especially as used in smoking, is attributed many cases of mouth cancer. Tobacco in itself may produce irritation either



Fig 142—Sarcoma of maxillary sinus. A Before surgery. B after surgery. The patient died of pneumonia eight years later without a recurrence of the lesion.

These growths are as a rule radioresistant and neither roentgen ray nor radium will have a curative or inhibitory effect, in most cases such therapy only delays operative treatment until it is too late to be of value to the patient. The contrary is true of carcinoma which is often cured or retarded by radiation therapy.

through smoking or other modes of use, but pipes may be responsible for the injury at the point of the original lesion. It is questionable that tobacco is responsible although nicotine may be. Broders' investigation of smokers and nonsmokers throws some doubt on tobacco playing any role in the production of cancer.



Fig 143—Epithelioma of the lip.

CARCINOMA, CANCER AND EPITHELIOMA—Malignant lesions of this type are found widely distributed in the oral cavity. They may originate in any region of the mouth or may be found in the mouth after they originate in contiguous tissue, especially in the nose, maxillary sinus and pharynx. If the point of origin is in the oral cavity, a history with some features of minor importance may be given by the patient, but more frequently the history is vague and un-

Epithelioma of the tongue may appear as a tumor or simply as ulceration, more often the latter. The differential diagnosis of lesions on the tongue may be difficult from a clinical examination alone. An examination of scrapings of the ulcer may reveal tubercle bacilli, in which case examination or roentgen ray of the chest will confirm or exclude a diagnosis of tuberculosis. Serological tests will usually exclude or confirm the presence of specific disease.

Although in the case of chancre negative results may be given in the first week a darkfield



Fig 144—Extensive epithelioma of the lip

In tertiary lesions or in primary ones that have been present more than a few days serological tests will eliminate or confirm the diagnosis of syphilis. It must be kept in mind that the lesion may be carcinomatous even though found in a tuberculous or syphilitic patient or even in one having both diseases as noted by Dr Joseph C Beck.

If still in doubt *biopsy* may be resorted to but the consent of the patient to an operation should be obtained before the section is made and the operation should not be delayed after the pathologist reports malignancy. In a biopsy the type of malignant growth should be noted in accordance with Broders' index since the kind of treatment or its extent may be dependent on the type. This is not only advisable but



A



B

Fig 145 Epithelioma of the lip. A Before operation. B after operation 5 years later.

examination of serum from the lesion will show spirochetes. Epithelioma is differentiated by its indurated irregular border and by biopsy.

DIAGNOSIS OF MALIGNANT GROWTHS—Cancerous lesions on cheeks, alveolus, lips, and tongue are, as a rule, easily diagnosed by the experienced but may be confused with other ulcerations of simple character, with tuberculous lesions, or with syphilitic lesions, either primary or tertiary. In tuberculosis the lesion is probably secondary to pulmonary disease, which can be readily diagnosed by a competent internist or by roentgen ray examination. If the lesions are syphilitic, scraping will show spirochetes on darkfield examination. In case of a primary lesion the history would probably tell the story, as chancre will only have been present a few days while a cancerous lesion will have been producing symptoms for weeks or months.



Fig 146—Epithelioma of the gum involving the maxillary sinus.

imperative. The types are graded from type 1 the least malignant to type 4 the most malignant.

nant Many tumors show different grades in different sections, hence much care must be exercised by the pathologist in consultation with the clinician in diagnosis

TREATMENT OF MALIGNANT GROWTHS—In small lesions of the lip, cheeks, and tongue

crustinate, however, and must be persuaded that their life chances and the cosmetic results are better if treatment is instituted early

If radiosensitive, the tongue may be treated with radium or roentgen ray and the lymph nodes should be irradiated, but if radioresist-



Fig 147.



Fig 148

Fig 147 —Leukoplakia of the tongue—removal with radium and electric cautery

Fig 148 —Leukoplakia of the tongue with carcinomatous change The patient was a heavy smoker and a syphilitic

radiation is frequently curative, especially in the less malignant types The kind of treatment also depends upon whether there is metastatic involvement of the lymph nodes and upon the extent of the lesion Roentgen-ray examination of the chest may show that metastatic involve-

ment, which can be determined after a very few treatments, operation should not be delayed The amount of tissue to be removed depends upon the judgment of the surgeon, but our best results have followed the removal of one-half of the tongue, either intra orally, or by the

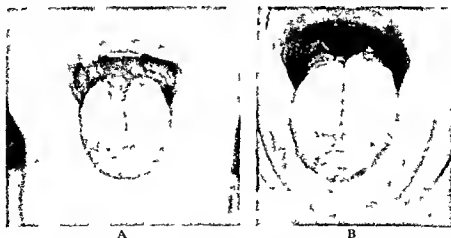


Fig 149 —A Carcinoma of the tongue (the patient was eighteen years of age), B twenty years later

ment is already present Which nodes are involved depends on the location of the primary lesion, whether on the lip, tongue, mandible, or maxilla Early diagnosis and operation immediately after the diagnosis promises the best chance for recovery Patients are prone to pro-

Kocher method, from below The latter is preferable when removing the entire tongue or when lymph nodes are to be removed at the same operation

When the alveolar process of the mandible is involved it may be possible to remove the



Fig. 150.—A, Carcinoma of the tongue; B, same patient five months later. This patient was alive and well seven years later.



Fig. 151.—A, Specimen of half of the tongue; B, patient one year after operation. This patient died of metastatic tumor of the lung two years after operation.



Fig. 152.—Carcinoma of face and maxillary sinus. A, External appearance; B, oral lesion.

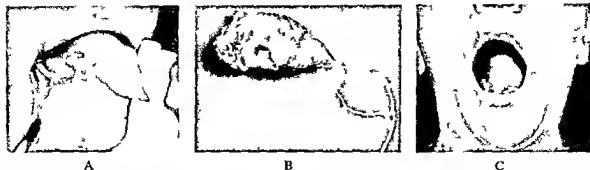


Fig. 153—A, Epithelioma of the tongue which was radioresistant to both radium and roentgen rays B One-half of tongue removed C, No local recurrence after four and one half years but lymph node in neck showed slight enlargement which has receded under roentgen ray treatment

involved portion by using bone forceps and cauterizing with high frequency current. Many good results were formerly obtained with soldering copper and an electrically heated cautery (the Percy cautery). This may devitalize the bone which will sequestrate and should be removed within about three months.

Cancer of the maxilla may be complicated by involvement of the maxillary sinus in which case it will be necessary to exenterate this cavity and possibly the nasal cavity on the involved side. The use of electricity in the form of a high frequency current is the most valuable. It may be necessary to remove portions of the wall of sinus, either involved or uninvolved bone, with rongeur forceps in order to gain access to and destroy all involved soft tissue.

Postoperative Treatment—Postoperative radiation is advisable and may be by screened radium, radon, or roentgen therapy. The postoperative care consists in cleansing and careful examination for possible recurrence. Some of these patients may require plastic procedures, especially if the face is involved or sloughing takes place after removal of the growth. In many cases, only a portion of the palate is involved, this may be replaced by a well constructed denture. In facial defects it may also be advisable to use a prosthetic appliance.

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DISEASES OF THE SALIVARY GLANDS

The salivary glands include the parotid gland, weighing between 14 and 28 gm., the submaxillary gland, weighing about 8 gm., and the sublingual gland, weighing 2 gm. The parotid duct (duct of Steno or Stensen), about 7 cm. long, runs a fingerbreadth below the zygoma, over the masseter muscle, to an orifice opposite the second upper molar. The submaxillary duct (duct of Wharton), about 5 cm. long, has a narrow orifice on a small papilla at the side of the frenum of the tongue. The sublingual gland has from eight to twenty excretory ducts (ducts of Rivinus), some of which join to form the larger sublingual duct (duct of Bartholin), which usually joins the submaxillary duct, while others (Walther's ducts) open separately on either side of the frenum. The parotid secretion is watery but rich in ptyalin. The submaxillary and sublingual secretions are viscid from the presence of mucin. Dryness of the mouth follows removal of the parotid glands, while the submaxillary and sublingual may be extirpated with little functional disturbance.

Anomalies—Congenital anomalies of the salivary glands are rare except for changes in the number and situation of the orifices of the submaxillary and sublingual glands

Traumatism—Closed or open wounds of the submaxillary or sublingual glands usually heal without marked reaction or fistula formation Should an external fistula form, it may be closed by the application of nitrate of silver or other caustic, or by excision and suture, provided the channel from the gland into the mouth is not obstructed For a persistent fistula of the parotid, a new duct may be established into the mouth, as will be described For a submaxillary or sublingual fistula the gland may be removed through an incision placed beneath and medial to the mandible, where the scar will be inconspicuous

Sialodochitis—Sialodochitis, or inflammation of the salivary duct, is usually associated with sialadenitis, or inflammation of the salivary gland It follows the entrance of foreign bodies into the duct, infections of the mouth and pharynx, and conditions associated with dryness and unusual bacterial proliferation within the mouth, as during continued fevers or after abdominal operations The orifice of the duct is red, pouting, and pus or turbid saliva may be expressed, while the corresponding salivary gland is tender and enlarged Treatment includes removal of foreign bodies and the eradication of any infection of the mouth or pharynx The orifice of the duct may be dilated or divided for better drainage, and the duct irrigated with a weak (1 500) permanganate, nitrate of silver (1 500, to 1 1000), or boric acid solution

Foreign Bodies—Foreign bodies found in the submaxillary duct include calculi, cereal grains, splinters of wood, seeds, bristles, and bits of feathers In glassblowers and players of wind instruments, air may enter and distend the salivary ducts, and exude in froth on pressure Symptoms due to the presence of foreign bodies may be slight or there may be violent salivary colic on mastication of food, as associated with swelling and tenderness of the salivary gland The tissues about the orifice of the duct are red, inflamed, and there is an associated discharge of turbid mucus or mucopus Calculi often may be seen through the thin overlying mucous membrane of the floor of the mouth or may be palpated by one finger within and others outside the mouth or demonstrated

by roentgenogram Usually salivary calculi are pale yellow and a few millimeters in thickness, but may reach a diameter of 3 cm They may consist of phosphate or carbonate of lime, with bacteria and organic material, and usually are found in men of middle age, commonly in the submaxillary duct, rarely in the submaxillary gland or in the parotid or sublingual gland or duct The foreign body may be removed by dilating or incising the orifice of the duct, or by direct incision into the duct or gland

Stricture or Stenosis of Salivary Ducts.—This may be due to injury, operation, inflammation, or neoplasm From stenosis the gland first enlarges and later tends to atrophy The condition is diagnosed by the introduction of probes and by sialography Treatment is carried out by gradual dilatation with probes For complete stenosis no treatment is required unless the gland remains swollen and painful, and then a submaxillary or sublingual gland may be excised, or for parotid involvement a fistula may be established by a seton into the mouth, as described for salivary fistula

Salivary Fistula—This results from (1) injury, (2) abscess, (3) ulceration and necrosis, as from noma, tuberculosis, or syphilis, or, rarely, (4) as a congenital defect Fistulas emptying on the skin of the face or neck are troublesome, those emptying within the mouth rarely require treatment Symptoms consist in the free discharge of saliva from the opening, especially on eating, with local irritation and eczema of the skin The differential diagnosis between a salivary fistula and a lymph fistula is determined by the character of the fluid, the influence of food and mastication upon the flow, and by sialograms made after injection of lipiodol into the duct and fistula

Glandular fistulas usually close spontaneously or after cauterization with silver nitrate, excision and suture, or plastic operation If these measures fail, complete excision of the submaxillary or sublingual gland is indicated Roentgenization to reduce the salivary secretion is rarely to be advised Duct fistulas of the parotid gland may occur from the buccal, masseteric, or glandular portion of the duct, usually with single, occasionally with multiple, openings which may discharge several hundred cubic centimeters of saliva every twenty-four hours Permeability of the buccal orifice is first determined by the introduction of probes or by injection of methylene blue solution If the

peripheral segment of the duct is open and the external opening contiguous, the fistulous opening may be closed by cauterization and pressure, by liberating and transplanting the proximal end to the buccal mucous surface, or by end-to-end anastomosis over a strand of alloy steel wire or silkworm gut, the end of which is brought through the normal orifice of the duct. The external opening is then treated

portion of the fistula, if large, should be excised and accurately closed with sutures. The silk thread seton, with the knotted end in the mouth, should be left in place from four to six weeks or more, and removed through the mouth. For glandular parotid fistula, when other measures fail, the salivary secretion may be arrested by ligating the central end of the duct, which, however, often produces an undesirable severe

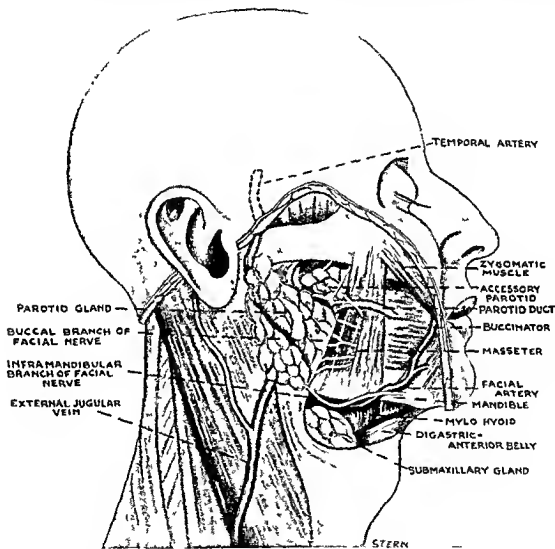


Fig 154—Salivary glands

by excision and suture. If the duct is impermeable an opening may be established from the fistulous tract into the mouth by means of a heavy silk thread, the ends of which are carried by a needle from the external fistula and brought through the mucous membrane about 5 mm apart, and tied (seton), or a small rubber tube may be introduced from the fistula through the mucous membrane and anchored for seven to ten days by a suture. In either case the external

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Sialadenitis—In sialadenitis the parotid gland is usually affected.

Acute Parotitis — **PAROTITIS EPIDEMICA** (MUMPS, SPECIFIC PAROTITIS)—This affection is characterized by self-limited, nonsuppurative, bilateral infection and enlargement of the parotid glands, usually occurring in the young,

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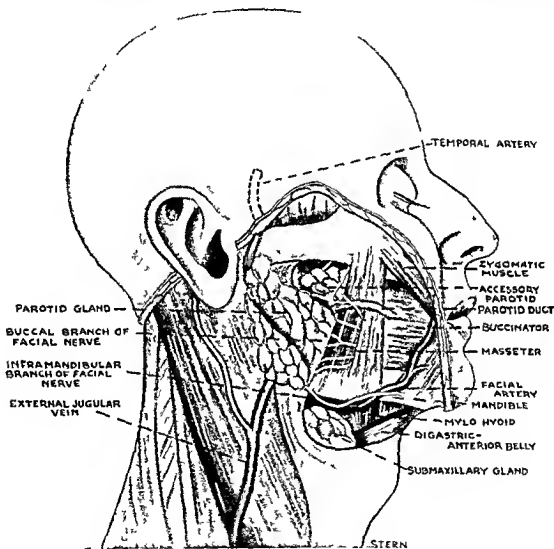


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possibly caused by a filterable virus. It is frequently complicated by orchitis, oophoritis, mastitis, or vulvitis and is occasionally followed by permanent dulness of hearing or, in the male, by sterility.

PAROTITIS PURULENTA (PAROTID BUBO, ACUTE SUPPURATIVE PAROTITIS)—This occurs (1) after stomatitis, salivation, suppuration of adjacent lymph nodes, or other infections in or about the mouth, (2) complicating a general infection, such as typhoid, smallpox, scarlet fever, or pyemia, (3) secondary to abdominal operations, usually developing five to seven days after an operation upon the gallbladder, appendix, intestine, or female genitals. The infection, usually unilateral, generally staphylococcic but occasionally streptococcic, may be metastatic or by contiguity, ascending through the ducts and favored by the dryness and bacterial overgrowth in the mouth. The symptoms include swelling, fever, tenderness and pain, increased by mastication, followed later by redness of the skin and fluctuation which may be difficult to detect owing to the thick overlying fascia. Turbid fluid or pus may be expressed from the duct. Diffuse phlegmon, facial paralysis, hemorrhage, septicemia, meningitis, or purulent mandibular arthritis may occur as a complication. The mortality varies from 10 to 43 per cent.

Prophylactic treatment consists in oral hygiene, removal of carious teeth and infected tonsils or adenoids, frequent moistening and cleansing of the mouth, and the use of chewing gum after abdominal operations or during general infections. With the onset of swelling, hot moist compresses of 2 per cent aluminum acetate or saturated magnesium sulfate solution, or a thick layer of yellow oxide of mercury ointment covered by cotton should be applied. Roentgen ray or radium irradiation has been largely used in the early stages to prevent suppuration, but may be ineffective. Penicillin or sulfonamide, given early, may arrest the process. When suppuration is evident, incision and drainage by Hilton's method, the incision being made parallel with and just anterior to the ear or below the angle of the jaw or in other situation where the scar will not be conspicuous, are employed, and yellow oxide ointment is applied and its use continued until healing occurs. If the process is very extensive an incision may be made from the zygoma along the anterior margin of the ear, and then curved

about the angle of the jaw. When the anterior border of this incision is retracted forward nearly the entire parotid gland is exposed. By puncturing the capsule of the gland in a number of places between the nerve fibers, with a pointed hemostat which is then opened, many points of drainage may be established and facial paralysis will be avoided. Wet antiseptic dressings are applied and continued until suppuration has ceased. When the infection has subsided the wound may be sutured, leaving little disability or scarring.

Chronic Parotitis—This may be caused by (1) obstruction of the parotid duct by foreign bodies, calculi, or stricture, (2) infectious foci in the mouth or pharynx, (3) poisoning by mercury, lead, copper, iodide, or opium or from uremia, (4) blood borne infection, or (5) tuberculosis. The latter cause is very rare, as is syphilis, which chiefly involves the parotid, with gummatous deposits, necrosis, salivation, and difficulty in talking and eating. Actinomycosis also rarely affects the parotid primarily. The symptoms of chronic parotitis include pain, tenderness, salivary colic on eating, salivation or dry mouth, salty or other taste, trismus, and a red parotid orifice from which turbid, purulent saliva or gas (acerocele of the parotid) may be extruded. Exacerbations often occur, last from three to ten days, and may recur at intervals of weeks or months. The diagnosis is determined by microscopic examination of secretion expressed from the duct and by sialograms. The treatment may be systemic, with elimination of mineral or other poisons. The foci of infection, stones or other foreign substance should be removed. Massage is often of benefit. Secretion may be stimulated by fruit acids or gum chewing. The ducts may be irrigated with iodized oil, or they may be dilated or incised and 2 to 3 cc. of saline solution or 2 per cent mercurochrome or other antiseptic instilled. Improved drainage with recovery may follow slitting of a constricted orifice of the duct, and suture of the edges of the duct to the cheek. If an abscess forms external drainage by Hilton's method should be used. Penicillin therapy may prove beneficial.

Sialodochitis Fibrinosa (Kussmaul)—This occurs in adults and is manifest by sudden swelling of one or both parotids, pain, trismus, and at times fever. It is relieved after hours or days by extrusion of a plug of mucus followed by

saliva Treatment consists in the administration of iodide of potassium or sodium

Mikulicz's Disease.—This disease is characterized by a symmetric and usually progressive enlargement of the lacrimal and salivary glands, with replacement of the glandular tissue by lymph cells It involves the parotid, submaxillary, sublingual, lacrimal, palatine, Blandin and Nuhn's, labial, and buccal glands as well as the spleen, which may enlarge gradually without pain or evidence of active inflammation It usually occurs in persons between twenty and forty years of age, who have paroxysms of enlargement lasting from a few hours to several weeks, and the condition may become stationary or recede after a febrile disease or treatment Xerostomia (dryness of the conjunctiva and mouth) and at times salivary lithiasis and narrowing of the ducts may accompany the deformity Disfiguring enlarged glands, except the parotid, may be excised Treatment may consist in a trial of arsenic and potassium iodide therapy Some improvement after roentgenization has been reported

Tumors—Benign Tumors.—Benign tumors constitute 83 per cent of tumors of the salivary gland Lipomas originate in the interlobular connective tissue and may be difficult or easy to enucleate Fibromas, myxomas, and angiomas are very rare The latter may grow rapidly Lymphangioma produces a prominent, compressible, or fluctuating mass which may be translucent and subject to acute inflammatory attacks The occurrence of adenoma is rare Papillary adenocystoma lymphomatousum is a benign, painless, slowly growing, firm, encapsulated, ovoid or lobulated, purple gray tumor with a smooth, finely granular, often cystic surface on section It is 2 to 6 cm in diameter and arises from branchiogenic heterotropic epithelium of the eustachian tube or salivary gland It usually develops after middle life, on the outer surface of the parotid or other salivary gland Cysts, congenital or acquired, quite rarely involve the parotid glands, and may be single or multiple The contents may be serous, mucoid, hemorrhagic, or the degenerative products of a necrotic tumor Diagnosis may be made by microscopic examination of aspirated contents Ranula, a transparent, thin-walled cyst of the floor of the mouth, with clear mucoid contents, is apparently due to obstruction of one of the smaller salivary ducts If punctured or incised the cyst usually refills

Benign tumors should be enucleated if solid, with great care to avoid injury to the branches of the facial nerve The incision described for exposing the suppurating gland may be used Through and beneath the capsule, the incision should run parallel with the fibers of the facial nerve, and blunt enucleation should be used if possible The angiomas or lymphangiomas may be sclerosed by intravascular hypodermic injections of a 33 to 50 per cent quinine urea solution, which, however, may interrupt conduction in nerve fibers Aspiration, which may be repeated, may be employed in benign cystic conditions, enucleation for cystic mixed tumors For ranula, treatment consists in the introduction of a seton to establish a permanent opening which may not succeed, or the excision of the anterior wall of the vesicle

Mixed Tumors.—A mixed tumor, the common salivary gland tumor, possibly an embryoma or teratoma derived from the branchial arches, may contain areas resembling cartilage, muscle, bone, a sarcoma, endothelioma, or carcinoma It usually develops in the parotid gland, especially in its retromandibular, pre-, sub-, or postauricular areas The epithelial cells form strands, diffuse masses, or fill alveoli and are found in association with the various mesoblastic portions The predominance of certain elements may suggest a chondroma, sarcoma, or carcinoma Mixed tumors occur rarely in the tongue, submaxillary, sublingual, or minor glands, but occasionally develop in the buccal mucosa, tonsillar region, palate, lips, or orbits, and may appear in individuals of any age but especially in those between twenty and forty, and in men They are more frequent on the left side than on the right and may be first noticed after an injury The growth is irregularly nodular, firm, but may have cystic areas It is usually encapsulated, movable, painless, and not attached to the cheek Only 11 per cent show malignant change The diagnosis is indicated by the presence of a circumscribed or lobulated tumor mass in the parotid gland, of long duration, without invasive characteristics, pain, or sensitiveness The histologic diagnosis may not be dependable

As a rule the mixed tumors or those containing cartilage are radioresistant Treatment consists in intracapsular enucleation through an incision along the anterior border of the ear or around the posterior border of the ramus and angle of the jaw The subcutaneous dissection

should follow the lines of the branches of the facial nerve, which should be protected carefully, and the tumor carefully enucleated from between the nerve fibers, as by a curet, and tincture of iodine applied by small swab to the capsule only. To avoid facial palsy the operation should be performed by an experienced surgeon. With clear evidence of malignant condition, an *en bloc* extirpation of the parotid and adjacent lymph nodes and contiguous tissue is indicated. The ensuing facial palsy may be improved by subcutaneous transplantation of strips of the temporal muscle, of fascia or silk threads to support the angle of the mouth and eyelids. From a long vertical incision, extensions may be brought over the mastoid or anteriorly to expose the entire gland. The facial nerve may be exposed from its deep position in front of the mastoid, and if the branches are not infiltrated by the tumor they may be liberated and held aside to permit the entire parotid to be removed in sections.

Malignant Tumors—Sarcoma as a pure tumor is rare in the parotid. Carcinoma is rather rare in the parotid, occurring as a scirrhous growth which is hard and infiltrative. Encephaloid cancer is soft, rapidly growing, and highly malignant. Carcinomas usually grow rapidly, as a rule are nodular, firm, infiltrative, adherent, and as they progress cause pain, facial palsy, and finally fixation of the overlying skin.

Adenocarcinoma, squamous and epidermoid carcinoma, and myxochondrocarcinoma have been reported but usually have been confused with the mixed tumors. The growth, which for a time is nodular, firm, nonadherent, and encapsulated, later may grow rapidly and infiltrate adjacent structures, with associated pain radiating to the shoulder and ear, facial paralysis, and ulceration of the skin. The sialogram shows incomplete and irregular filling of the duct system with puddling or diffusion of the lipiodol in adjacent tissues (Blady).

Treatment is carried out by radical, early *en bloc* excision when possible, with removal of tributary lymph nodes. The implantation of radium or radon seeds and heavy external irradiation in the majority of cases are ineffective and serve but to increase the local irritation and pain.

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DISEASES OF THE PHARYNX AND FAUCES

ACUTE DISEASES OF THE PHARYNX AND FAUCES

The differential diagnosis of acute sore throat is important, for the life of the patient depends upon prompt recognition and treatment of sore throat due to diphtheria or agranulocytic angina, while the future health of the community as well as of the individual depends upon the early recognition and treatment of sore throat due to primary and secondary syphilis.

The following conditions are to be considered in a case of acute sore throat: streptococcus infections of the throat, peritonsillar abscess, retropharyngeal abscess, diphtheria, Vincent's angina, primary and secondary syphilis, agranulocytic angina, infectious mononucleosis, acute leukemia, thrush, herpes simplex.

Streptococcal Infections of the Pharynx—The great majority of cases of acute sore throat represent an infection with a streptococcus, usually hemolytic, sometimes nonhemolytic, rarely viridans. Four types of hemolytic streptococcal throat infections are distinguished, depending upon the local manifestations, the systemic manifestations, or the mode of infection. These are acute pharyngitis, acute follicular tonsillitis, scarlet fever, and septic sore throat.

Acute Pharyngitis—This is an acute inflammation of the mucous membrane of the pharynx, usually of infectious origin. Most often the etiologic agent is the hemolytic streptococcus, but it may be the nonhemolytic streptococcus, a pneumococcus, the influenza (Pfeiffer's) bacillus, or a filterable virus. These organisms are frequently found in the pharynx of normal individuals. They became pathogenic either as the result of a new virulent strain being introduced, or because the local and general resistance has been lowered owing to a virus infection (common cold or influenza), fatigue, chilling, or, occasionally, an allergic reaction to a food or inhalant.

The streptococcus infection in the mucous membrane is analogous to the streptococcus infection in the skin in erysipelas. The inflammation begins in the nasopharynx and extends gradually downward to the hypopharynx, or vice versa. The mucous membrane of the pharynx may appear almost normal, or show varying degrees of diffuse redness, frequently with

edema of the uvula. The lymphoid nodules on the posterior pharyngeal wall, in the nasopharynx (the adenoid), and in the lateral walls of the pharynx (the lateral pharyngeal bands) are red and swollen, and in severe infections there will be scattered, rarely confluent, spots of yellowish-white exudate on them. Adenitis of the posterior and anterior cervical glands is frequently present.

The symptoms are dryness, scratchiness, and soreness in the pharynx, with pain on swallowing, malaise, and slight to moderate fever depending upon the severity of the infection, all come on rather acutely and reach a climax within a day or two. The severity of the symptoms varies greatly with the resistance of the patient, the virulence of the infecting organism, and the size of the infecting dose.

In making a diagnosis simple acute pharyngitis, due usually to the hemolytic streptococcus, is not likely to be confused with sore throat from other causes, but if there is exudate and the symptoms came on gradually over several days diphtheria must be ruled out by culture, if the sore throat lasts for days and weeks secondary syphilis must be ruled out by a blood serologic test, if ulcerations develop agranulocytosis must be ruled out by a white blood cell count.

The frequency of complications is directly proportional to the severity of the infection. Complications in the majority of cases are confined to local extension of the mucosal infection, either upwards to cause an acute otitis media, a rhinitis or a sinusitis, or downwards to cause a laryngitis, tracheitis, bronchitis, or pneumonia. Extreme edema of the uvula and suppurative of the cervical glands may occur.

Systemic complications due to blood borne streptococci or their toxins, such as acute nephritis or acute rheumatic fever, may result from sore throats having relatively mild local manifestations. As a rule, however, systemic complications occur chiefly in the very severe infections, and besides nephritis and rheumatic fever include septicemia, meningitis, peritonitis, erysipelas, pneumonia, thrombophlebitis of a cervical vein, and pyemia.

In planning the treatment it is important to remember that the great majority of cases of acute pharyngitis even though due to the hemolytic streptococcus run a benign and self-limited course with rest in bed, avoidance of exposure to cold air, and a hot saline gargle.

every few hours. Ordinary infectious precautions should be observed to protect others, and 10 grains (0.6 gm) of aspirin (acetylsalicylic acid) should be given every four hours for pain and discomfort. A gargle consisting of 10 grains (0.6 gm) of aspirin dissolved in 2 tablespoonfuls (30 cc) of hot water is comforting for the sore throat. For annoying dryness of the mucous membranes steam inhalations with compound tincture of benzoin, 1 teaspoonful (4 cc) to a kettle of water kept boiling on an electric plate by the bedside, are of value. The value of painting the throat is questionable, but swabbing the pharynx with 10 per cent argyrol at the onset may be tried. Silver nitrate (10 per cent) carefully applied on a tightly wound applicator to the tip of any large inflamed lymphoid nodules may be of value.

The local use of sulfonamides has been recommended, but the value of their local application for pharyngitis is difficult to prove. Nevertheless, it seems a logical procedure, provided the drug reaches the inflamed areas and is not irritating. Sulfadiazine powder insufflated through a powder blower every three or four hours is probably the best means of local application.

The systemic use of a sulfonamide should be reserved for the more severe infections with high fever, or with ear, sinus or pulmonary complications, or with blood borne septicemic complications. Sulfadiazine is to be preferred as being less toxic and as effective as sulfathiazol or sulfanilamide. The average adult dose is 15 grains (1 gm) of sulfadiazine every six hours until the symptoms and fever subside, then, to prevent recurrence, the dose is decreased gradually for several more days. With each dose of sulfadiazine, sodium bicarbonate, 1 teaspoonful (4 gm), should be given to alkalinize the urine and minimize the precipitation of crystals in the urinary tract. Sufficient fluids should be given to maintain a minimum output of 1000 cc of urine in twenty four hours, and to minimize the precipitation of crystals. The dose of sulfadiazine for children is about 2 grains (0.1 gm) per pound per day, a relatively larger dose than for adults due to the more rapid excretion of the drug by children.

A white blood cell count and urinalysis should always precede the administration of a sulfonamide and should be repeated every two or three days during administration of the drug, with immediate withdrawal of the drug in the

event of leukopenia or hematuria. If the fever does not abate within forty eight hours on full doses of the drug, the organism is probably sulfonamide resistant and there is little advantage in continuing the medication. It should be remembered that the drug itself may cause a high fever, especially around the fifth to the seventh day of medication, or immediately upon its administration in a person sensitized by previous administration. A skin rash (including a red sore throat), mental confusion, hemolytic anemia, headache, anorexia, nausea, and vomiting are other toxic manifestations. The drug should be stopped if any of these occur. In general, the toxic manifestations are in proportion to the total amount of the drug given. Maximum doses of the drug to maintain a blood level of 10 to 20 mg per 100 cc of blood are indicated only for the most serious complications such as meningitis. In such cases, to get an immediate effective blood level, an initial dose of 30 to 60 grains (2 to 4 gm) may be given.

The routine use of a sulfonamide in full therapeutic doses or even in small doses, for ordinary acute pharyngitis of average severity, must be condemned for these reasons: (1) In mild and moderately severe uncomplicated pharyngitis even when due to the hemolytic streptococcus neither the duration nor the severity of the attack is demonstrably lessened by the sulfonamides. (2) The risks from the drug equal or exceed the risks from the infection. Most of the fatal results from sulfonamide administration have been due to anuria, either from an acute nephritis or a deposition of crystals in the kidneys. Therefore, oliguria or hematuria are to be watched for closely. (3) The period of debility from the drug with fatigue and weakness over many weeks may exceed the debility from the infection. (4) Sensitization of the patient to the drug may prevent its use for a more serious subsequent infection.

In addition to watching the patient for toxic manifestations watching the urine for oliguria or hematuria, and watching the blood for leukopenia or anemia, it is important that ultraviolet light to the throat or skin be withheld during sulfonamide medication, as this increases the likelihood of a skin rash. The administration of epsom salts (magnesium sulfate) and other sulfur compounds is also contraindicated during sulfonamide medication, to avoid a serious and perhaps fatal sulfhemoglobinemia.

For patients who are sensitized to the sulfonamides, or in infections that do not respond to the sulfonamides, where the infecting organism is a virulent hemolytic streptococcus, and there is a high degree of toxicity, the use of human convalescent scarlet fever serum is valuable. The average adult dose is 20 cc. of serum intramuscularly or intravenously. Immuno transfusions, that is the use of blood from a donor recently recovered from a hemolytic streptococcus infection, is also valuable in severe infections.

In recurring attacks of acute pharyngitis a focus of infection, in the form of an infected adenoid, an infected tonsil or tonsil remnant, infected enlarged lateral pharyngeal bands, or a chronic suppurative sinusitis, should be searched for and removed. If no focus can be found a house-dust or food allergy should be suspected, searched for and treated or removed. Local immunization of the nasal and pharyngeal mucosa by an intranasal vaccine spray, as advised by Cannon and Walsh, may be tried. The use of vitamins and oral "cold" vaccine or stock "cold" vaccines by hypodermic injection have been of doubtful value. Maintaining the general bodily resistance, by avoidance of overheating, chilling, and fatigue, is helpful. Finally, contact with persons who have an acute pharyngitis should be avoided if possible.

Acute Follicular Tonsillitis (Acute Lacunar Tonsillitis)—This is an acute inflammation of the faucial tonsils with patches of exudate over the openings of the crypts. It is essentially the same infection as acute pharyngitis, except that the inflammation is predominantly in the tonsils. It is nearly always due to the hemolytic streptococcus. A previous chronic tonsillitis is a predisposing factor.

The mucous membrane lining all of the crypts of the tonsil is involved in the infection, a much larger area of mucosa in a smaller region thus being involved than in simple pharyngitis, with correspondingly greater local and systemic manifestations. The tonsils are much enlarged and reddened, with small patches of yellowish-white fibrinous and purulent exudate over the mouths of the crypts. Occasionally ulcerations in the depths of the crypts result in thrombi in the vessels at the base of the crypts. It is easy to see how such a process results in systemic infection. The anterior cervical glands below the angle of the jaw are enlarged and tender. In very severe infections there may be local necro-

sis of the surface of the tonsils, with areas of ulceration. The patches of exudate in such cases may coalesce and form a membrane resembling diphtheria.

The symptoms of acute tonsillitis are sore throat, with difficulty in swallowing, malaise, and fever, all of abrupt onset and generally greater than in simple acute pharyngitis.

In determination of the diagnosis, if there is increasing exudate, diphtheria must be ruled out by a culture. The membrane of tonsillitis is rarely confluent, is thin and superficial, and does not stick, whereas the diphtheritic membrane is thicker and usually confluent and adherent. Both scarlet fever and infectious mononucleosis should be considered in a case of tonsillitis.

The same systemic complications may occur as in acute pharyngitis, especially acute nephritis and acute rheumatic fever, with or without cardiac involvement. Peritonsillar abscess (quinsy) may follow in a few days. Septic thrombophlebitis of the internal jugular vein is a rare complication, manifested by a high swinging fever, chills, a positive blood culture and tenderness along the vein.

The treatment is the same as for acute pharyngitis. The application of 10 per cent or 20 per cent silver nitrate to the surface of the tonsils is said to diminish the inflammation and shorten the attack. The cotton applicators should be tightly wound, and all excess solution pressed out to prevent the silver nitrate from running down into the larynx.

Recurring attacks of acute tonsillitis are an indication for tonsillectomy.

Scarlet Fever—Scarlet fever is an acute pharyngitis or tonsillitis caused by a particular strain of hemolytic streptococcus the toxin of which produces a skin rash. It is transmitted by contact with a person with scarlet fever, or with a carrier harboring the streptococci of scarlet fever in his throat. Scarlet fever occurs predominately in children, usually in epidemics, with an incubation period of two to six days after exposure.

The pharyngitis and tonsillitis of scarlet fever are identical with ordinary pharyngitis or tonsillitis except that, as a rule, the infection in scarlet fever is rather severe and the pharynx and tonsils show a greater degree of inflammation. The characteristic skin rash appears in twenty-four to forty eight hours, lasts three to seven days, and consists of a diffuse redness

with many minute red points most marked on the neck, trunk, and flexor surfaces of the extremities. The face may be flushed but is free from the punctate rash found elsewhere on the body, which helps to differentiate it from the rash of measles. Desquamation is characteristic of the rash of scarlet fever, it includes the skin of the palms and soles and begins soon after the rash fades.

The symptoms consist in an abrupt onset of sore throat and fever, with malaise, prostration, and vomiting, followed in twenty four to forty eight hours by the characteristic skin rash.

Diagnosis is made when the characteristic rash appears, but should be suspected before the rash appears in an acute pharyngitis or tonsillitis during a scarlet fever epidemic.

The complications are the same as in acute pharyngitis, but more frequent and more severe.

The treatment is the same as for acute pharyngitis, plus the use of human convalescence scarlet fever serum or antitoxin for severe toxicity, and sulfadiazine for the ear sinus and systemic complications. The patient should be isolated from those who have not had scarlet fever.

Septic Sore Throat—This is an epidemic form of acute pharyngitis traceable to milk contaminated with pathogenic hemolytic streptococci. From a human source, the streptococcus enters the udder of the cow through the lactiferous ducts during milking and for many days or weeks the milk may continue to be loaded with pathogenic hemolytic streptococci. Epidemics of sore throat always occur from drinking raw milk, never from pasteurized milk.

Septic sore throat is an acute pharyngitis of more than average severity, due to the huge size of the infecting dose, with inflammation, swelling, and edema of the pharynx and tonsils often with exudate, and with marked cervical adenitis.

The symptoms consist in an acute onset of high fever, prostration, and sore throat occurring after a brief incubation period (two to four days) after drinking raw milk.

Complications are the same as in acute pharyngitis, but especially frequent, occurring in a fourth of the persons afflicted with suppuration of the cervical lymph nodes, septicemia, and dissemination of the streptococci to the kidneys, joints, peritoneum, meninges, heart, and skin.

The diagnosis is made when a severe pharyn-

gitis occurs in epidemic form traceable to a supply of raw milk contaminated with pathogenic hemolytic streptococci.

Treatment of septic sore throat is the same as for acute pharyngitis. The mortality from any future epidemics should be lessened with the use of scarlet fever antitoxin or human convalescent scarlet fever serum, and the sulfonamides.

Only pasteurization of milk can prevent future epidemics, since attacks have been traced to the most carefully kept herds.

Streptococcus Viridans Pharyngitis—This is a superficial indolent ulceration due to infection with *Streptococcus viridans*. In this type of pharyngitis a circumscribed shallow ulceration, covered by a membrane, is usually located on the tonsil or palate.

The only symptom is a sore throat, with little or no constitutional reaction, tending to persist for several days to a week or more.

The condition resembles a large herpes simplex, or suggests Vincent's angina. The absence of constitutional symptoms, the absence of Vincent's spirilla, and a positive culture of *S. viridans* make the diagnosis.

Gentian violet in a 2 per cent aqueous solution applied to the ulcer a few times usually results in rapid healing.

Acute Uvulitis—Acute inflammation of the uvula, with redness and edema, may accompany an acute infectious pharyngitis or an acute allergic reaction. If the edema is sufficient to embarrass deglutition or respiration it may be reduced by the local topical application of adrenalin (1:1,000). Occasionally amputation of the tip of the uvula will be required. This is accomplished, after the topical application of 10 per cent cocaine solution for anesthesia, by grasping the tip with a tonsil tenaculum or with a hemostat and then cutting off with scissors a few millimeters of the tip. This permits the edematous fluid to escape, and the subsequent cicatrization reduces the size of the uvula.

Abscesses of the Pharynx and Fauces.—Peritonsillar Abscess (Quinsy Sore Throat)—This is an abscess outside the faucial tonsil, between it and the constrictor muscles of the pharynx, in the connective tissue of the tonsil capsule. It is caused by an acute infection in a tonsil whose crypts have become sealed off as the result of previous infection. An abscess occurs in the depth of the tonsil which ruptures into the connective tissue of the tonsil capsule, usually at

the upper pole of the tonsil. There is redness and marked swelling of the soft palate on the side of the abscess, with displacement of the uvula toward the opposite side. The tonsil itself is displaced toward the midline, the anterior cervical glands below the angle of the jaw are enlarged and tender, the constrictor muscles of the pharynx are inflamed and there is very marked pain and difficulty in swallowing. The pterygoid muscles are inflamed and trismus is present, and the deep cervical muscles are inflamed, the neck being held rigidly inclined toward the side of the abscess.

The *symptoms* consist in an acute pharyngitis or tonsillitis followed in a few days by increasing soreness and pain on one side, with extreme difficulty and pain in swallowing, drooling, trismus, and the head held stiffly inclined slightly toward the side of the abscess. Stridor due to pharyngeal obstruction may occur. Despite the very distressing pain and the patient's evident misery there is, as a rule, less fever and toxemia than from an ordinary follicular tonsillitis.

Complications are very unusual. Downward extension, edema of the glottis, and thrombophlebitis of a large vein in the neck, have been described.

In determining the *diagnosis*, a unilateral severe tonsillitis, simulating a peritonsillar abscess, may be ruled out if the uvula is displaced, since this does not occur in simple tonsillitis. Acute leukemia, chancre, gumma, or carcinoma of a tonsil may also simulate a peritonsillar abscess, but the relatively acute onset and rapid development of the latter, with the characteristic displacement of the uvula should make differentiation relatively easy. Rarely, a carotid artery aneurysm may simulate a peritonsillar abscess.

Local heat in the form of hot saline throat irrigations every hour or two is the most effective *treatment* until the abscess is localized and ready to be opened. A douche can or hot water bottle is filled with hot normal saline solution, a glass medicine dropper is placed in the end of the rubber tube and the stream of saline is directed against the painful area by the patient while he holds his head forward over a basin, with the mouth open. When there is evidence of a localized accumulation of pus in the form of a rounded bulging, with or without fluctuation, the abscess should be incised and drained.

Incision should be made over the region of the greatest bulging or fluctuation. This is most

often at the intersection of a horizontal line on a level with the soft palate and a vertical line level with the anterior faucial pillar. Owing to the marked swelling and displacement the location of the latter must be determined at the junction of the anterior pillar with the tongue. Preliminary infiltration of the point of incision with 1 or 2 per cent novocaine containing 8 drops of adrenalin to the ounce (0.5 cc of adrenalin to 30 cc of novocaine) will lessen the pain. A vertical incision a half inch (1 to 1.5 cm) long is made through the mucous membrane and a narrow artery forceps is plunged in between the layers of the tonsil capsule and spread. If pus is not encountered the hot throat irrigations should be resumed for a day or two until the abscess is more localized, when it should again be incised over the area of greatest bulging and fluctuation.

Tonsillectomy for drainage of an acute peritonsillar abscess has sometimes been practiced, but with occasional serious and even fatal complications (septicemia) so that this procedure is inadvisable.

Later, after the abscess has been drained and the infection has subsided tonsillectomy should always be done, for the pathologic condition responsible for the abscess makes a recurrence probable. The optimum time for tonsillectomy after peritonsillar abscess is three to four weeks after drainage. At this time the local and general immunity of the patient is at a high level, and the scar tissue forming in the healing abscess has not yet become so dense and sclerosed as to render the tonsillectomy difficult.

Retropharyngeal Abscess—Retropharyngeal abscess may be defined as an accumulation of pus between the posterior pharyngeal wall and the bodies of the vertebrae. The abscess is formed by the suppurative of one of the lymph nodes found in the upper posterior wall of the nasopharynx, secondary to an acute pharyngitis. In older children these lymph nodes have generally disappeared so that acute retropharyngeal abscess is essentially a disease of infancy, more than half the cases occurring in the first year of life. The pus lies in the retropharyngeal fascial space between the constrictor muscles of the pharynx in front and the prevertebral fascia behind. The abscess may extend downward, or laterally to the parapharyngeal space, but the abscess generally localizes and "points" in the upper posterior pharynx. Extension to the mediastinum, while possible, is rare.

Following an upper respiratory infection an unexplained fever in an infant or young child, with loss of appetite and difficulty in swallowing, should arouse the suspicion of retropharyngeal abscess. As the abscess works downward the airway will become narrowed with labored breathing and the stridor characteristic of airway obstruction in the pharynx. The child may hold his neck more or less rigidly to keep open the airway.

Death due to respiratory obstruction is not infrequent, and may occur when a mouth gag is inserted to examine or open the abscess, or if the abscess suddenly ruptures spontaneously flooding the pharynx and larynx with pus. Hemorrhage, sudden and profuse, usually means that the abscess has extended laterally and has eroded the internal carotid artery. The initial profuse bleeding ceases when the blood pressure drops and clotting occurs, but a recurrence of the hemorrhage may be expected as the pus invades and softens the blood clot, and the blood pressure recovers. While the child usually survives the first such profuse hemorrhage, the second or the third will very likely be fatal. Therefore, prompt ligation of the common carotid artery on the side of the abscess must be done to prevent recurrent and probably fatal hemorrhages.

The diagnosis of retropharyngeal abscess is easily missed because the infant is too young to complain of his throat, and casual inspection of the pharynx may fail to detect the uniform pushing forward of the posterior pharyngeal wall. A lateral roentgenogram of the neck is the most reliable aid in diagnosis and should be made in all doubtful cases. In the presence of an abscess there will be a marked increase in the soft tissue shadow between the pharyngeal airway and the bodies of the vertebrae. Protrusion of the vertebrae from malformation or simply from twisting the neck, may on inspection of the pharynx simulate a retropharyngeal abscess, but palpation will serve to differentiate the doughy abscess from the hard bony protrusion. If there is dyspnea, forcible opening of the mouth with a gag or palpation of the posterior pharyngeal wall should be done cautiously and only when suction and a Mosher lifesaver are at hand, in case of sudden respiratory obstruction.

As soon as retropharyngeal abscess is diagnosed, treatment should consist in immediate drainage. With suction available, the child is

placed on a table with a folded blanket under his shoulders to lower the head. A mouth gag is gently and carefully inserted and opened, and a vertical incision is made with a short bladed scalpel, or with a longer scalpel wrapped with adhesive except for the tip, over the point of greatest swelling. The knife may be passed along the index finger which is placed on the abscess as a guide. If pus does not appear a closed narrow artery forceps should be introduced, pushed in deeply and spread. Suction, or quickly turning the child onto his stomach with the head and shoulders hanging over the edge of the table, as soon as pus appears, will prevent aspiration. The incision should be spread once a day until no more pus escapes. As a rule recovery is rapid in acute retropharyngeal abscess once drainage is established. In the event of persistent fever sulfadiazine in appropriate dosage according to the weight of the child, should be given, and a search made for other causes of the fever.

Diphtheria—Diphtheria is an acute communicable disease due to the diphtheria bacillus. It is transmitted by direct contact with individuals afflicted with the disease, or carriers who harbor the bacilli in the pharynx.

The bacilli in the mucosa of the pharynx produce a toxin which destroys the surface epithelium, with a resulting exudation of serum that coagulates and forms a membrane. The membrane, dirty gray in color, is usually (but not always) confluent, it tends to spread beyond the tonsils onto the soft palate, it is adherent and is removed with difficulty, it has well-defined margins and a characteristic odor described as "mousy." The cervical lymph nodes are enlarged and tender.

The soluble toxin produced by the bacilli causes a myocarditis (the most frequent cause of death) and paralysis of the muscles of the soft palate, muscles of accommodation and, less often, the extraocular muscles, the muscles of the pharynx, diaphragm, and extremities. Paralysis occurs in 15 to 20 per cent of the cases.

After an incubation period of two to five days there is a gradual onset of sore throat, low grade fever, and increasing toxemia. The pharynx is first red, then the membrane appears usually beginning on the tonsils and spreading steadily to the pillars, uvula, and, sometimes, to the posterior pharyngeal wall. The membrane may spread to or may occur primarily in

the nose or larynx, rarely in the trachea and bronchi

The adherent confluent membrane, the gradual onset of symptoms as contrasted to the acute onset in tonsillitis, the high degree of toxicity with the comparatively low grade fever, are all characteristic. The *diagnosis* is confirmed by finding the Klebs Loeffler bacilli on smear and culture, the swab should be rubbed against the membrane, and then on Loeffler's medium, and examined after twelve to eighteen hours of incubation

Edema of the throat, obstruction, paralysis, and myocarditis are possible *complications*. Laryngeal obstruction due to laryngeal involvement frequently requires intubation or a tracheotomy. Paralysis, especially of the palate, comes on in the second or third week of convalescence and lasts two or three weeks. Myocarditis, with acute circulatory failure or heart block, is the usual cause of death. Marked edema of the throat and neck producing a "bull neck" occurs in severe infections.

Antitoxin administered as early as possible and in adequate dosage (10,000 to 50,000 units are the average dose for an adult) is the most important and effective *treatment* in neutralizing the toxin. *Every hour of delay in making the diagnosis and giving the antitoxin adds to the danger to the patient's life.* Isolation, bed rest, and glucose given intravenously complete the treatment.

Vincent's Angina (Trench Mouth)—This is a specific lesion in the mouth or pharynx due to a spirillum, a fusiform bacillus, and an aerobic streptococcus in symbiosis. Usually it is contracted by direct contact with infected individuals, especially when it occurs in epidemic form (trench mouth). It may also arise from organisms already present in the mouth or tonsils as a result of poor oral hygiene and lack of sufficient vitamin C. A localized ulceration with a ragged border and crater, filled with a grayish slough or membrane, occurs especially on the tonsils or gums. Beneath the membrane are granulations that bleed easily.

The *symptoms* are sore throat or mouth, malaise, slight to moderate fever, a fetid breath, and enlarged and tender cervical nodes. Hemorrhage from the ulcer is frequent.

Complications are rare except for a tendency to chronicity of the infection in the gums.

Diagnosis is made by the demonstration of the fusiform bacillus and the spirillum in a

smear made from the ulcer, stained with gentian violet.

The *treatment* usually consists in local applications. These are generally very effective in promptly arresting a progressive ulceration, and in effecting a cure. Since the infecting organisms are anaerobic, an oxidizing agent such as sodium perborate—1 teaspoonful (4 gm) in $\frac{1}{2}$ cup (60 cc) of hot water—should be used as a mouth wash and gargle three or four times a day. Arsphenamine or neoarsphenamine—10 grains (0.6 gm) dissolved in 4 tablespoonfuls (30 cc) of glycerin—should be applied to the ulcer on a cotton applicator once or twice a day until the lesion has cleared. Rarely will it be necessary to give arsphenamine or neoarsphenamine intravenously. Any deficiency in vitamin C in the diet should be corrected.

Recurrent attacks of Vincent's infection of the tonsils indicate a tonsillectomy.

Syphilis of the Throat—Chancere of the Tonsil—This is a primary syphilitic infection of the tonsil. Infection with the *Spirochaeta pallida* is transmitted by direct contact with a person with a primary or secondary syphilitic lesion, frequently by kissing a person with secondary lesions of the mouth, but it may be by drinking from a cup or glass that has just been used by such a person.

A diffuse infiltration of the connective tissue with round cells and epithelioid cells and an acute obliterative endarteritis causes an indurated swelling usually of one tonsil, with an indolent ulceration on the surface of the tonsil. The "tonsillar" lymph node below the angle of the jaw is enlarged and indurated.

A persistent sore throat lasting more than a week with a unilateral enlarged indurated ulcerated tonsil, suggests a chancre. Later the skin rash, generalized lymphadenopathy, and slight fever and malaise of secondary syphilis occur.

Diagnosis is made by a darkfield demonstration of the *S. pallida*. The blood serologic reaction is not yet positive in early chancre.

Prompt antisymphilitic therapy should be instituted with precautions against spreading the disease to others.

Secondary Syphilis of the Throat—Syphilitic sore throat occurs in the secondary stage of syphilis. The typical mucous membrane lesion of secondary syphilis is the "mucous patch" consisting of slightly elevated, dull red areas with a round or oval or kidney shaped superficial erosion, a shiny, dirty, waxy, or pearly

surface, and fairly well-defined borders. The mucous patch usually occurs in the tonsil fossae, on the tonsils or pillars, or on the buccal and inner surfaces of the lips. However, there may be merely a reddened pharynx without visible mucous patches.

The *symptoms* are a persistent sore throat, with generalized lymphadenopathy, a skin rash, mild malaise, and slight to moderate fever, occurring between the sixth and twelfth week after the appearance of the chancre.

A persistent sore throat lasting more than a week, with generalized lymphadenopathy, with or without a skin rash, should arouse a suspicion of secondary syphilis. The *diagnosis* is made by the positive blood serologic test, the *S. pallida* may be demonstrated in smears made from the mucous patches.

Prompt antisyphilitic therapy should be given with precautions against infecting others.

Agranulocytic Angina—This disease is characterized by sore throat, usually with rapidly spreading necrotic lesions in the tonsils, pharynx, and mouth, associated with a marked depression or complete absence of granulocytes (polymorphonuclear leukocytes) in the circulating blood. A toxic depression of the bone marrow occurs which may be due to infection or to a drug, especially amidopyrine (pyrimidin) and the sulfonamides. Individual idiosyncrasy (allergy) to the drug appears to be a determining factor, the occurrence of agranulocytosis being more frequent when large doses of the drug are given over a long period of time, or when the patient has been sensitized by previous dosage.

The bone marrow shows a marked depression or absence of leukopoietic activity. The pharynx, at first reddened, with or without membrane formation, develops necrotic ulcers, the margins of which are undermined with very little inflammatory reaction in the tonsils, soft palate, uvula, gums, or tongue. Ulcerations may also occur in the larynx, esophagus, stomach, bowel, vagina, urinary bladder, trachea, and bronchi.

Marked prostration is the most common and characteristic *symptom*. The fever is slight in the chronic cases, marked in the acute fulminating cases. *Jaundice* may occur. The white blood cell count is low (2000 or less) and the differential count shows a marked reduction or complete absence of polymorphonuclear leukocytes (granulocytes).

Hemorrhages may occur from the ulcerations. Anemia occurs in the chronic cases. Death occurs in as high as 75 per cent of the severe acute cases, and may occur within a few days of the onset of the sore throat.

Diagnosis is made from the white blood cell count and differential count, showing the granulocytopenia. In every pharyngitis with marked prostration and toxemia, particularly with ulcerations, a white blood cell count and blood smear should be made. Acute leukemia with leukopenia may be confused with agranulocytosis, but the presence of many immature leukocytes in the former will serve to differentiate the two.

The first step in the *treatment* is the immediate discontinuance of all drug therapy, as this may remove the etiologic agent. One or 2 gm of pentose nucleotide administered intravenously daily to stimulate the bone marrow appears to be of value. Repeated blood transfusions may tide the patient over until his bone marrow begins to function again.

Infectious Mononucleosis—This disease is an infection occurring frequently in epidemics and characterized by lymphadenopathy and a typical blood picture. The causative organism is not known. Infection is apparently by contact, with an incubation period of about seven days.

General hyperplasia of the lymph nodes occurs throughout the body. An acute pharyngitis or tonsillitis often with membrane formation is frequent. Enlargement of the spleen is common. The characteristic blood picture is a lymphocytosis giving a total blood count of from 10,000 to 20,000 with an increased proportion of lymphocytes. These lymphocytes are larger than normal, with a blue, "foamy" appearing cytoplasm and often an eccentric nucleus.

The *symptoms* are malaise, headache, moderate fever, generalized lymphadenopathy, and frequently a persistent sore throat. The fever and enlarged, often tender, lymph nodes may persist for weeks, occasionally for months.

The sore throat may appear like an ordinary pharyngitis or tonsillitis, but its persistence and the generalized lymphadenopathy should suggest infectious mononucleosis. The *diagnosis* may be made from the blood picture. A positive heterophile antibody test (sheep cell agglutination) confirms the diagnosis. A falsely positive serologic test for syphilis may occur, and with

the sore throat and generalized lymphadenopathy secondary syphilis may be simulated

Gastro intestinal symptoms, jaundice, and central and peripheral nerve involvement, with increased cells in the spinal fluid, sometimes occur as complications. Recovery is the rule.

The treatment is purely symptomatic.

Acute Leukemia—Acute leukemia is characterized by a permanent increase in the leukocytes of the blood with hyperplasia of the leukopoietic tissues and immature white cells in the blood. The cause is unknown.

The tonsils may be swollen and markedly enlarged suggesting a peritonsillar abscess. Ulceration of the tonsils is frequent. Generalized lymphadenopathy, including the cervical glands, is usual.

The symptoms are sore throat, fever, hemorrhages from the mucous membranes, and a rapid anemia.

Diagnosis is made from the blood picture—a leukocytosis of 20,000 to 30,000 or higher with many immature forms.

Treatment is symptomatic. No effective treatment is known. The condition is always fatal.

Thrush—Thrush is a stomatitis or pharyngitis characterized by adherent small white patches on the mucosa due to a fungus. A species of *Monilia*, or varieties of *Oidium*, acquired by direct contact or by air, is the etiologic agent.

Small white flakes adherent to the mucous membrane, usually leaving a bleeding point when removed, occur especially on the faucial and lingual tonsils and pharyngeal lymphoid nodules of adults and cheeks and lips of infants. Except for these white patches the mucosa shows very little inflammatory reaction.

Sometimes a slight to moderate sore throat without fever or other constitutional signs is present. Often there are no symptoms. Microscopic examination of one of the white flakes will show the mycelial threads.

There are no complications.

Gentian violet in a 1 or 2 per cent aqueous solution applied on cotton applicators every day or two is usually an effective therapeutic agent.

Herpes Simplex (Cold Sores, Fever Blisters).—The disease is an acute infection caused by a virus and characterized by small superficial watery blisters on the skin or mucous membrane. The etiologic agent is a virus, which may remain implanted in the tissues of certain indi-

viduals and give rise to recurring herpes as the result of certain stimuli such as allergic reactions, some infectious diseases, and menstruation. A small vesicle is formed in the skin or mucous membrane, surrounded by a narrow zone of hyperemia. Rupture of the vesicle on the skin results in the formation of a crust, on the mucous membrane it results in a small, round, sharply defined ulcer. Multiple herpetic vesicles may coalesce to form an irregular ulceration in the mucous membrane.

The herpetic lesion itself rarely produces constitutional symptoms, the only symptom being pain and soreness rather sharply localized to the region of the herpetic lesion.

The sharply localized, painful ulceration, with a narrow border of erythema, is not likely to be confused with other types of ulceration of the mucosa of the mouth and throat, except where the herpetic lesions are multiple and coalesce. Herpes is found most often on the tongue and lips and buccal mucosa, but may be on the soft palate.

Silver nitrate (10 per cent) applied on a tightly wound cotton applicator to the ulcer usually results in almost immediate relief from the pain and in prompt healing.

CHRONIC DISEASES OF THE PHARYNX AND FAUCES

Simple Chronic Pharyngitis—The disease is a chronic inflammation of the mucous membrane of the pharynx without infiltration or ulceration. Allergy to a food, to an inhalant, especially to house dust, or to smoking, is a frequent cause of chronic pharyngitis. Infection in a sinus, in the adenoid, or in the lateral pharyngeal bands may cause a chronic pharyngitis. Atrophic rhinitis resulting in dryness and crusting in the nasopharynx, is frequently a cause.

The pharyngeal mucosa may show considerable redness, with hypertrophy of the superficial lymphoid nodules, and still not be a source of symptoms. The appearance of the mucosa must be interpreted in relation to the symptoms as within the limits of normal, or as definitely pathologic. In chronic pharyngitis the usual changes are diffuse redness, enlargement and redness of the superficial lymphoid nodules, especially in the lateral pharyngeal bands, and dryness and crusting, especially in the nasopharynx.

Dryness, scratchiness, a sense of irritation, at times with soreness and slight discomfort on swallowing, are the symptoms.

Simple chronic pharyngitis must be differentiated from chronic granulomas of the pharynx.

Discovery and removal of the etiologic factor is of primary importance in the treatment. Smoking should be omitted for at least two weeks as a therapeutic test. Where the lateral hands are inflamed and enlarged and appear to be the source of the symptoms 10 per cent silver nitrate should be applied to them at weekly intervals. Surgical removal of the lateral hands is indicated when the silver nitrate applications fail to reduce the inflammation. This is best done with a ring punch under local novocaine anesthesia. Occasionally the lingual tonsil will be the seat of a chronic inflammation. If the local application of 10 per cent silver nitrate does not relieve the symptoms the hypertrophied and inflamed lingual lymphoid tissue may be removed surgically, by means of a Nyles lingual tonsillotomy. For the annoying dryness and crusting in the nasopharynx normal saline solution should be sniffed up into the nose twice daily. A nasal spray of 3 per cent alcohol and 6 per cent glycerin in normal saline solution will stimulate the nasal secretions and counteract the dryness.

Chronic Granulomas of the Pharynx—Granulomatous pharyngitis is a chronic infiltrative inflammation caused by certain specific microorganisms. The tubercle bacillus, the spirochete of syphilis, the leprosy bacillus, or the fungi causing actinomycosis, blastomycosis, or coccidioid granuloma, may be responsible.

The histologic changes in chronic granulomas of the pharynx vary somewhat with the specific causative organisms, but consist essentially in chronic inflammation of the subepithelial connective tissue with a dense infiltration with round cells, the formation of granulation tissue, and areas of necrosis, including the epithelium. With special stains the specific causative organism may be found in the tissue.

Tuberculosis of the pharynx like tuberculosis of the larynx, is always secondary to an active pulmonary tuberculosis. The pharyngeal mucosa shows irregular, worm eaten, shallow ulcerations containing pale granulations. Pain on swallowing is usually marked. *Lupus* of the pharynx is associated with lupus of the skin of the face and like the latter is characterized by superficial ulcerations which tend to heal cic-

trize, and recur. *Gumma* of the pharynx begins as a painless swelling which later ulcerates and finally heals with extensive scarring, often leaving a perforation of the palate or pillars. *Leprosy* causes nodules and ulcerations in the pharynx that heal with extensive loss of tissue and scarring. *Actinomycosis* produces a firm painless soft tissue swelling, followed by ulceration and suppuration. The purulent discharge contains small yellowish pellets, "sulfur granules," composed largely of the typical ray fungus. *Blastomycosis* produces ragged superficial ulcers with soft granulating floors and a purulent discharge. *Coccidioid granuloma* of the pharynx consists of diffuse granular thickening and infiltration of the soft palate and pillars with superficial erosions of the epithelium.

Chronic sore throat, with more or less pain on swallowing, is the main symptom of chronic granuloma of the pharynx.

A blood serologic test for syphilis, a chest plate for pulmonary tuberculosis, a biopsy, and microscopic study of a smear from the lesion, stained for the specific organisms, are the means of diagnosis.

Management of tubercular pharyngitis consists of treating the pulmonary lesion first, and giving analgesics for the pain in the pharynx. Ultraviolet light locally may be of benefit. The usual antisyphilitic therapy for tertiary syphilis is used for gummas. Leprosy is treated with chaulmoogra oil. For the chronic fungus infections iodides in large doses are given.

Chronic Diseases and Anomalies of Uvula—*Elongation of the Uvula*—This condition rarely produces symptoms. However, the uvula may be of sufficient length to cause gagging and perhaps coughing. Treatment consists in amputation of the tip as described for "acute uvulitis".

Bifid Uvula—This is a congenital anomaly due to incomplete fusion in embryonic life. No treatment is required.

Tumors of the Uvula—Benign papilloma occurs not infrequently on the uvula, and if large may be excised, if small it should be left alone.

Chronic Tonsillitis—This is a chronic infection of the faucial tonsil. A severe acute tonsillitis such as occurs in scarlet fever frequently leaves the tonsils chronically infected. In some cases the tonsils became the seat of a chronic infection without the history of an acute tonsillitis. The hemolytic streptococcus is probably the most frequent etiologic agent.

The tonsils are chronically inflamed and usually enlarged owing to hyperplasia of the lymphoid follicles. The inflammation is localized in the tonsils and the anterior pillars where they overlie the tonsils, the redness of these structures being in contrast to the paler color of the soft palate and posterior pharyngeal wall. The crypts of the tonsils may contain an abnormally large amount of cellular (caseous) debris. Small ulcerations may be found in the depths of the crypts and scar tissue may close off some of the crypts resulting in small retention abscesses.

The *symptoms* of chronic tonsillitis are local and systemic. The *local* symptoms of chronic tonsillitis are (1) recurring attacks of acute follicular tonsillitis, (2) frequently recurring attacks of mild sore throat localized in the tonsil region, (3) chronic sore throat localized in the tonsillar region, (4) chronic or recurring adenitis of the cervical gland below the angle of the jaw (the tonsillar lymph node), and (5) peritonsillar abscess. The *systemic* symptoms of chronic tonsillitis, due to absorption of bacteria or their toxins into the circulation, are malnutrition and loss of appetite, chronic fatigue, secondary anemia, and metastatic infection in the joints, muscles, nerves, bursae, inner ear, eye, and other regions, resulting in acute and chronic arthritis, myositis, neuritis, bursitis, labyrinthitis, and iritis. In these cases the tonsils act as a focus from which infection is disseminated to other parts of the body.

The *diagnosis* of chronic tonsillitis is made from the symptoms, and from the signs. The signs of chronic tonsillitis are localized redness of the tonsils and overlying anterior pillars, enlargement of the tonsils, an embedded position of the tonsils so that the anterior pillar may entirely conceal the tonsil from view until a blunt tonsil hook or pillar retractor is used to pull the anterior pillar laterally, causing the patient to gag and the embedded tonsil to stand out as a rounded swelling behind the pillar, an enlarged cervical gland below the angle of the jaw, frequently with tenderness of this gland, liquid pus that can be expressed from the crypts by pressure on the anterior pillar (the caseous debris found normally in the tonsil should not be mistaken for pus), excessive amounts of caseous debris in the tonsil crypts, and small yellowish retention abscesses in the tonsil.

Tonsillectomy is the only completely effective treatment for chronic tonsillitis. Palliative treatment in the form of roentgen ray therapy will

shrink down an enlarged tonsil but it is doubtful that the decrease in size will eliminate the chronic infection. The local application of 10 to 20 per cent silver nitrate to the surface of an inflamed tonsil at weekly intervals appears to decrease the inflammation. Evacuation of the crypts by pressure on the anterior pillars or by suction, using a rubber bulb and a special glass cap that fits over the tonsil, will temporarily remove excessive caseous debris. Electrocoagulation of the tonsils has been abandoned largely because of the great difficulty in completely removing all tonsillar tissue by this means without damaging the constrictor muscles and even the deep vessels of the neck, and because of the dense scar left covering any tonsil remnants preventing drainage from the crypts and therefore favoring systemic absorption as well as attacks of peritonsillar abscess.

Chronic Infection of the Adenoid—This is a chronic infection and inflammation of the pharyngeal tonsil, or adenoid, usually with an enlargement of this structure.

The adenoid normally reaches its greatest development in middle childhood, and atrophies and completely disappears after puberty. With chronic infection abnormal hypertrophy occurs and frequently persists into adult life. Chronic infection of the adenoid, as of the tonsil, is generally the result of an acute infection which fails to clear up. Histologically there is hyperplasia of the lymphoid follicles. Grossly the adenoid is enlarged for the patient's age, and shows varying degrees of inflammation. Retention abscess particularly in the midline (Thornwaldt's disease) may occur as the result of scar tissue following an operation (adenoidectomy) or a severe infection which has sealed off one of the fissures.

The *symptoms* of chronic infection of the adenoid are systemic and local. The *systemic* symptoms occur when the adenoid acts as a chronic focus of infection, and are the same as for chronic tonsillitis. The *local* symptoms of chronic infections of the adenoid are nasal obstruction and mouth breathing due to blocking of the airway by the enlarged adenoid, defective hearing due to blocking of the eustachian tubes by the enlarged adenoid, frequent and prolonged head colds, recurring attacks of acute suppurative otitis media, and chronic suppurative otitis media in cases where the infected adenoid keeps the eustachian tube infected.

Diagnosis is made by inspection, with a post-

nasal mirror, nasopharyngoscope, or, in roomy noses by anterior rhinoscopy after shrinking the inferior turbinates. In some children inspection is unsatisfactory and palpation must be resorted to. The child is placed on his back, his head is held firmly by one attendant and his hands are held by another. The physician stands at the head, places two or three wooden tongue blades between the left molar teeth (to protect his fingers), and introduces the right index finger gently behind the soft palate to the roof of the nasopharynx. This procedure is uncomfortable and momentarily painful, but if the child is warned beforehand that it will hurt for a moment he will usually forgive the doctor. A patient either child or adult, resents being hurt by surprise more than being hurt with fair warning.

Surgical removal (adenoidectomy) is indicated if there is sufficient adenoid tissue to reach the upper margin of the choanae as seen by posterior mirror rhinoscopy. Where there is less lymphoid tissue than this in the midline, and where the lymphoid hypertrophies extend into the fossae of Rosenmüller and over the torus tubarius, particularly following a well-done surgical adenoidectomy, roentgen ray or radium therapy of the nasopharynx is indicated.

TECHNIC OF TONSILLECTOMY AND ADENOIDECTOMY

Indications and Contraindications—The tonsils and adenoids should be removed when they are the seat of a chronic infection which produces or threatens to produce bodily injury greater than the dangers, disability, discomfort, and inconvenience of removal. Routine removal of tonsils and adenoids in children is to be condemned as entirely unnecessary, and as subjecting the child to the risks of surgery without any assurance of benefit. Moreover the probable protective function of Waldeyer's ring against infections entering through the nose and mouth may be impaired by removal of normal tonsils and adenoids. In adult life the tonsils have no function. There are no definite age limits for tonsillectomy, but weighing the dangers and disadvantages of operation against the probable benefits of removal tonsillectomy will rarely be indicated under three years of age and after sixty years of age.

Operation is *contraindicated* in the presence of an acute infection of any kind but especially an acute respiratory tract infection. Other con-

traindications are a blood dyscrasia such as hemophilia, purpura, leukemia, or a severe anemia, uncontrolled diabetes, cardiac disease or nephritis, at least until further study of the patient has been made, and enlarged thymus gland. To rule out the presence of one of these complications a history of recent acute infection or of an abnormal tendency to bleed should be obtained, a bleeding and clotting time determination and a urinalysis should be routine, and the patient's throat, heart, and lungs should be examined.

Choice of Anesthetic—For children, for adults with enlarged adenoids, and for excessively nervous adults who prefer to be asleep, a general anesthetic (ether) is used. A local anesthetic (novocaine) is preferred for adults.

Preoperative Measures—There is no satisfactory evidence that the routine administration of calcium, vitamin D, C or K, or other substances before operation is of benefit in decreasing bleeding. Fluoroscopic or roentgenographic study for enlarged thymus in children is routine in some clinics, but is not generally done unless there has been evidence of tracheal compression. The meal preceding the operation should be omitted. Preoperative medication for children under ten consists of atropine in appropriate dosage, 1/600 grain to 1/200 grain (0.1 mg. to 0.3 mg.), given hypodermically one hour before operation. For adults who are to have a general anesthetic, and for older children $\frac{1}{2}$ to $\frac{1}{4}$ grain of morphine according to size with 1/150 grain of atropine are given hypodermically one hour before operation. For adults who are to have local anesthesia, nembutal or some similar quick-acting barbiturate is given by mouth, the first dose of 1½ grains (0.1 gm.) one hour before and the second dose of 1½ grain (0.1 gm.) given just before operation.

Technic of Tonsillectomy under General Anesthesia—*Position of the Patient*—Because the most frequent serious complications of tonsillectomy under general anesthesia are pulmonary (atelectasis, pneumonia, lung abscess), owing probably to aspiration of blood and caseous debris from the tonsils, the hyperextended position of the head is preferable (Fig 155, A). In this position the larynx lies higher than the upper pharynx so that with suction to keep the pharynx free there is almost no possibility of aspiration of blood and detritus into the larynx and trachea. The operating table should have a headpiece that can be lowered

Anesthesia is induced with the patient lying flat, by nitrous oxide and oxygen, or by ethylene, or by cyclopropane, followed by ether. As soon as relaxation occurs a small roll 2 to 3 inches high made of a folded sheet is placed under the shoulders and the head of the table is dropped 4 to 6 inches. The Davis mouth gag with the small, medium, or large tongue blade according to the size of the patient, is then inserted and opened (Fig 155, B, C). Rubber tubing connects an ether vaporizer to a metal tube on the mouth gag. The handle of the mouth gag, held by the anesthetist who sits on the patient's right, should assume a vertical position. The surgeon sits on a stool at the head of the table with a head lamp for illumination, a suction tube over his shoulder, and the instrument table on his left.

Removal of the Adenoids—The adenoids are removed first so that bleeding from the nasopharynx will have stopped before the patient returns to bed. The index finger is gently inserted behind the soft palate to evaluate the size and distribution of the adenoids. The La Force adenotome, preferably a modification with the reversed curve (Fig 157, B) is inserted behind the soft palate with the blade opened, it is then pressed as far as possible forward against the posterior end of the septum, as well as against the roof of the nasopharynx, and is closed, removing the adenoid that lies in the midline (Fig 155, D). Above the protuberance of each eustachian orifice, in the fossa of Rosenmüller, there may now be palpated adenoid tissue which must be removed by rotating the adenotome to one side or the other so as to enter the corresponding fossa of Rosenmüller (Fig 155, E). It is important to keep the adenotome tight against the roof of the nasopharynx during this maneuver so that the cartilaginous protuberance of the eustachian tube will not be caught and injured by the adenotome. At the conclusion of the adenoidectomy the roof of the nasopharynx is felt as a smooth, hard concavity with no tags of soft lymphoid tissue against the septum, in the midline or in either fossa of Rosenmüller. A small gauze pack about $1\frac{1}{2}$ inch by 1 inch, tied with a length of tape, is now inserted and left in the nasopharynx with the tape hanging out of the mouth (Fig 155, F) until the conclusion of the tonsil operation.

Removal of the Tonsils—With the hyperextended position of the head, preferred because of the lessened incidence of pulmonary com-

plications, the guillotine methods are not applicable. Dissection and snare are used. The aim of the tonsil operation is to split the capsule of the tonsil so that the removed tonsil can be seen to be covered by a smooth, shiny, intact layer of connective tissue and the tonsil fossa will also be seen to be lined by smooth, shiny intact connective tissue. To keep within the layers of the capsule and out of the lymphoid tissue of the tonsil, on the one hand, and the constrictor muscle of the pharynx on the other, preserving a maximum of intact mucosa on the pillars, is the aim of the technic.

The tongue blade of the mouth gag is adjusted so that the tongue is pushed to one side, leaving the opposite tonsil well exposed (Fig 156, G). This tonsil is grasped with a curved tenaculum and moved slightly to and fro to demonstrate clearly the fold of the anterior pillar and the plica triangularis—the triangular fold of mucous membrane extending from the lower half of the anterior pillar down over the lower pole of the tonsil to the base of the tongue. (In the inverted position of the head the lower pole is up, but to avoid confusion the operation will be described as though the tonsil were being observed with the patient upright.) The first incision, made with the straight end of the tonsil knife (Fig 157) is through the mucous membrane of the plica triangularis parallel with and just behind the anterior pillar (Fig 156, G). At this point the tonsil capsule is well defined and easily split, so that when the tonsil is pulled out slightly with the tenaculum, the incision gapes, revealing the smooth, pearly gray capsule covering the tonsil. The curved end of the tonsil knife is now inserted deeply into the incision and is drawn along the posterior edge of the anterior pillar to the upper pole, cutting with the inside edge of the curved blade (Fig 156, H). The knife, still in the incision, is then pushed down the posterior aspect of the tonsil along the anterior edge of the posterior pillar, cutting with the outside edge of the curved blade (Fig 156, I). The upper pole of the tonsil, exposed by this single sweep of the knife, is further freed by using the outside curve of the blade pushed down between the upper pole and the fossa. The blunt Hurd dissector is now used to complete the elevation of the upper half to two thirds of the tonsil from the fossa, taking care not to dig into the constrictor muscle of the pharynx (Fig 156, J). Finally the wire snare is placed around the tonsil and the remaining

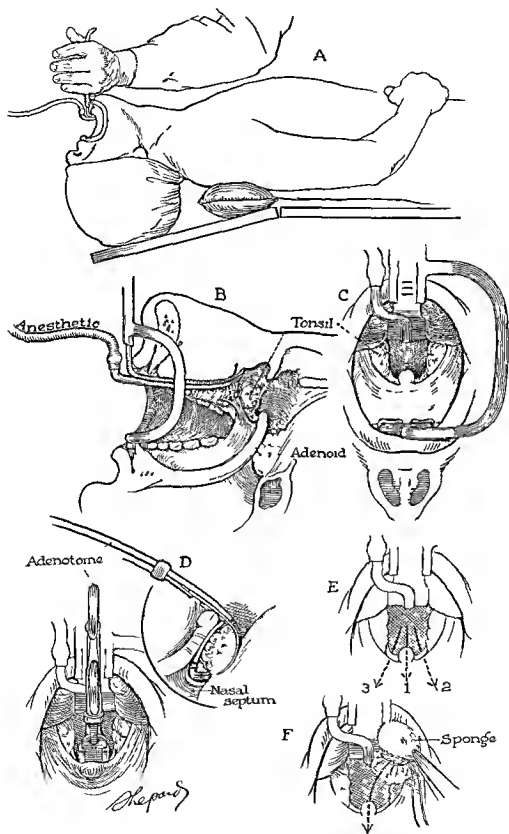


Fig 155 —Technic of tonsillectomy under general anesthesia

attachment of the lower pole is snared off slowly so as to slowly crush the blood vessels and favor their rapid sealing by blood coagulation (Fig 156 K)

Unless there is a large arterial spurter, the initial bleeding is not disturbed except to keep

ally profuse or prolonged, however, the bleeding point must be identified by cleansing the fossa by suction and grasped with a curved hemostat which may then be left in place while the second tonsil is being removed. A suture or tie is rarely required. If ligation of a bleeding

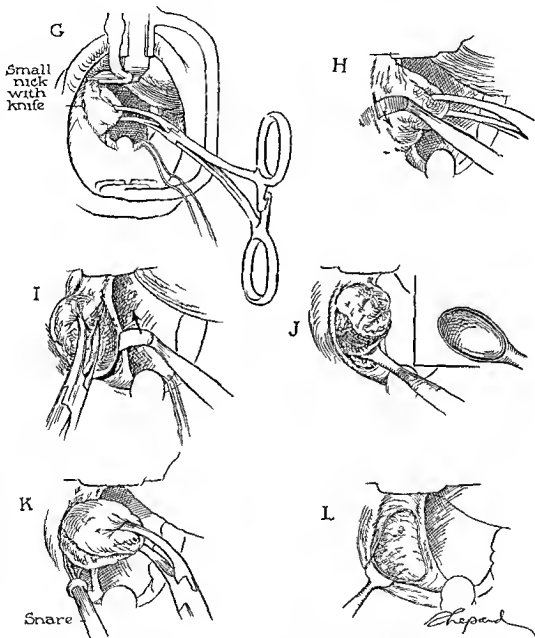


Fig 156—Technic of tonsillectomy under general anesthesia (cont nued)

the pharynx clear of blood with suction. It has been shown experimentally that sponging with gauze removes the tissue juices (thrombokinase) necessary to the normal process of blood coagulation and hemostasis, and actually prolongs the bleeding. Within a few minutes the bleeding usually subsides and stops. If unusu-

ally profuse or prolonged, however, the bleeding point must be identified by cleansing the fossa by suction and grasped with a curved hemostat which may then be left in place while the second tonsil is being removed. A suture or tie is rarely required. If ligation of a bleeding

nants, using the pillar retractor on the end of the Hurd dissector (Fig 156, L), any remnants being grasped with the tenaculum and removed with the snare. A nodule of lingual tonsil can often be seen at the lower pole and this must be grasped and snared off. The postnasal pack is then removed, and when all bleeding has stopped, the mouth gag is removed and the patient is sent back to bed. The anesthetic should be regulated so that the gag reflex returns before the mouth gag is removed and so that, if possible, the patient will empty his stomach by

of impending shock: rapid, thready pulse, cold clammy extremities, pallor, and restlessness. Any of these calls for immediate inspection of the throat for active bleeding. Water by mouth is permitted when the child is awake and emesis has ceased. Aspirin may be given for pain.

The child remains in the hospital over night, and in the morning, if his temperature is below 100° F, his color and pulse good, and there is no bleeding, he may return home. The first day at home is best spent in bed. Thereafter activity may be permitted short of fatigue and short of

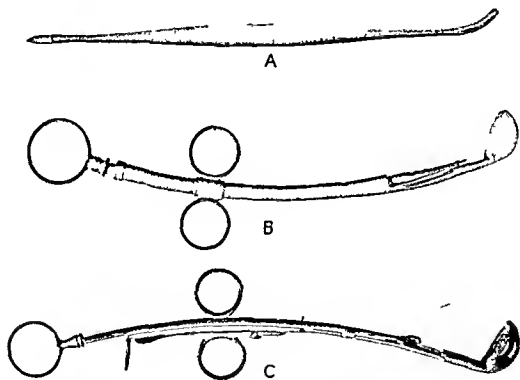


Fig 157 —A Shambaugh tonsil knife for use in the head hyperextended position. B Shambaugh adenotome for use in the head hyperextended position. C regular La Force adenotome.

emesis. This frequently eliminates the very uncomfortable postoperative retching and vomiting, and it decreases the possibility of a large loss of blood down the esophagus while the child is still asleep.

Postoperative Care—The child is placed in bed on his stomach with his head resting on his outstretched arm. He should be partially awake by the time he reaches his bed, and fully awake within an hour. He should be watched for any visible bleeding or vomiting of blood and for invisible bleeding, as evidenced by signs

of excessive exertion for ten more days. Most children will eat a full diet after the second day, but older children and adults will prefer soft foods, such as liquids, cooked cereals, milk, toast, custard, and ice cream. The acid of fruit juices will cause considerable pain, and so should be avoided. Older children who can gargle should use a teaspoonful of sodium perborate (flavored) in a glassful of warm water gargled after each meal for ten days. Aspergum, chewed, will alleviate much of the soreness and may be used freely.

Postoperative Complications—Hemorrhage occurs most often within the first twelve hours or about a week after operation, at which time the fibrinous exudate covering the cut surface separates, leaving granulations which in a few more days will be covered with new epithelium. Bleeding from a tonsil fossa is controlled by removing the clot carefully with a curved hemostat and small gauze sponges, and identifying and grasping the bleeding point with the hemostat. If there is diffuse oozing, tannic acid powder on a small gauze sponge moistened with 1:1000 adrenalin may be placed in the fossa to cause a superficial coagulation of the raw surface. Occasionally a severed blood vessel will retract beneath the surface and bleed into the tissues, resulting in a large hematoma beneath the tonsil fossa from which blood oozes in numerous places. Attempts to grasp the bleeding point are of no avail, the tissues infiltrated with blood being very friable. The best treatment in this troublesome type of bleeding is sedation with morphine, and prolonged pressure on the tonsil fossa with a sponge. It will greatly facilitate the procedure in any postoperative hemorrhage if morphine is given in appropriate dose to quiet the patient and lessen the pain, the morphine itself is valuable for shock.

Bleeding from the adenoid is statistically more dangerous than from the tonsil because much blood may be swallowed without any external evidence of bleeding, and because the bleeding point cannot be seen. Occasionally the use of the pillar retractor to lift up the soft palate will reveal a bleeding point which may be grasped with a hemostat. Usually, however, a postnasal pack must be inserted as follows. A small rubber urethral catheter is inserted through a nostril until it appears in the pharynx, where the end is grasped with a hemostat and pulled out of the mouth. One end of a string is tied to the end of the catheter, the other end of the string being tied around the postnasal pack made out of plain or iodoform gauze, loosely folded and tied into a pack about $1\frac{1}{2}$ inch long and 1 inch thick. The catheter is then pulled back through the nose, pulling the string after it. The catheter is then inserted into the other nostril and the procedure is repeated so that finally there is a string through both nostrils, coming out the mouth, and tied around a single postnasal pack. A third string should be tied to the pack so that when it has been pulled up into the nasopharynx there will remain one end of string out the

mouth by which the pack may later be removed. In placing the postnasal pack the palate must be pulled forward with a pillar retractor as the two strings through the nose are pulled on. It is well to insert the index finger into the nasopharynx to push the pack snugly into position. The two strings coming out the nose are now tied together across the front of the septum over a small piece of gauze to protect the skin. The postnasal pack should be removed within twenty-four hours to lessen the danger of infection of the middle ears through the eustachian tubes. If the bleeding recurs, the pack must be reinserted, preferably an iodoform pack which inhibits the growth of organisms and lessens the danger of otitis media.

Atelectasis is evidenced by fever, rapid respirations, cough, perhaps dyspnea and cyanosis. The physical signs are dullness on the affected side, with absent breath sounds. Fluoroscopy showing failure of the lung or lobe to aerate, with displacement of the mediastinum toward the collapsed side, together with elevation of the diaphragm on the collapsed side, confirm the diagnosis. The treatment is to move the patient and, if possible, have him sit in a chair, encourage coughing, have him lie on his good side, administer carbon dioxide to cause hyperventilation. If these measures fail, aspirate the occluding plug of mucus and blood bronchoscopically.

Pneumonia is evidenced by the same symptoms as atelectasis, but the breath sounds on the affected side are increased rather than absent, and fluoroscopy shows that the diaphragm is symmetrical, the mediastinum is in the mid line, and both lungs aerate equally.

Lung Abscess is evidenced by fever, cough, and the expectoration of a mouthful of foul pus, usually a week or two after operation. The diagnosis is confirmed by roentgenography. There is no recorded instance where lung abscess followed tonsillectomy in the head-hyperextended position.

Technic of Tonsillectomy under Local Anesthesia—*Anesthesia and Position of Patient*—Tonsillectomy under local anesthesia is done with the patient sitting upright in a chair, the surgeon sitting in front on a stool, with a head lamp for illumination. A sterile sheet is placed over the patient, and a basin in his lap which he holds with both hands (Fig. 158, A). If the patient gags excessively, the throat may be sprayed first with 2 per cent pontocaine. There

nants, using the pillar retractor on the end of the Hurd dissector (Fig 156, L), any remnants being grasped with the *tenaculum* and removed with the snare. A nodule of lingual tonsil can often be seen at the lower pole and this must be grasped and snared off. The postnasal pack is then removed, and when all bleeding has stopped, the mouth gag is removed and the patient is sent back to bed. The anesthetic should be regulated so that the gag reflex returns before the mouth gag is removed and so that, if possible, the patient will empty his stomach by

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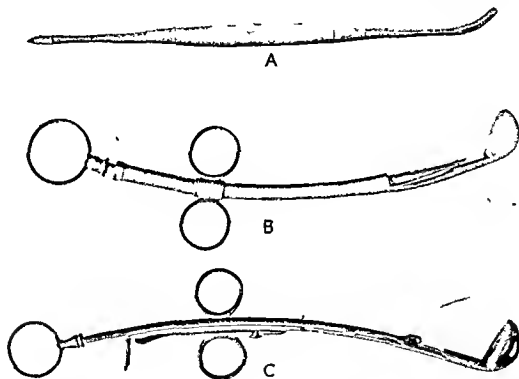


Fig 157—A Shambaugh tonsil knife for use in the head hyperextended position. B, Shambaugh adenotome for use in the head hyperextended position. C regular La Force adenotome.

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excessive exertion for ten more days. Most children will eat a full diet after the second day, but older children and adults will prefer soft foods, such as liquids, cooked cereals, milk toast, custard, and ice cream. The acid of fruit juices will cause considerable pain, and so should be avoided. Older children who can gargle should use a teaspoonful of sodium perborate (flavored) in a glassful of warm water gargled after each meal for ten days. *Aspergum*, chewed, will alleviate much of the soreness and may be used freely.

Postoperative Complications—Hemorrhage occurs most often within the first twelve hours or about a week after operation, at which time the fibrinous exudate covering the cut surface separates, leaving granulations which in a few more days will be covered with new epithelium. Bleeding from a tonsil fossa is controlled by removing the clot carefully with a curved hemostat and small gauze sponges, and identifying and grasping the bleeding point with the hemostat. If there is diffuse oozing, tannic acid powder on a small gauze sponge moistened with 1:1000 adrenalin may be placed in the fossa to cause a superficial coagulation of the raw surface. Occasionally a severed blood vessel will retract beneath the surface and bleed into the tissues, resulting in a large hematoma beneath the tonsil fossa from which blood oozes in numerous places. Attempts to grasp the bleeding point are of no avail, the tissues infiltrated with blood being very friable. The best treatment in this troublesome type of bleeding is sedation with morphine, and prolonged pressure on the tonsil fossa with a sponge. It will greatly facilitate the procedure in any postoperative hemorrhage if morphine is given in appropriate dosage to quiet the patient and lessen the pain, the morphine itself is valuable for shock.

Bleeding from the adenoid is statistically more dangerous than from the tonsil because much blood may be swallowed without any external evidence of bleeding, and because the bleeding point cannot be seen. Occasionally the use of the pillar retractor to lift up the soft palate will reveal a bleeding point which may be grasped with a hemostat. Usually, however, a postnasal pack must be inserted as follows. A small rubber urethral catheter is inserted through a nostril until it appears in the pharynx, where the end is grasped with a hemostat and pulled out of the mouth. One end of a string is tied to the end of the catheter, the other end of the string being tied around the postnasal pack made out of plain or iodoform gauze, loosely folded and tied into a pack about $1\frac{1}{2}$ inch long and 1 inch thick. The catheter is then pulled back through the nose, pulling the string after it. The catheter is then inserted into the other nostril and the procedure is repeated so that finally there is a string through both nostrils, coming out the mouth, and tied around a single postnasal pack. A third string should be tied to the pack so that when it has been pulled up into the nasopharynx there will remain one end of string out the

mouth by which the pack may later be removed. In placing the postnasal pack the palate must be pulled forward with a pillar retractor as the two strings through the nose are pulled on. It is well to insert the index finger into the nasopharynx to push the pack snugly into position. The two strings coming out the nose are now tied together across the front of the septum over a small piece of gauze to protect the skin. The postnasal pack should be removed within twenty-four hours to lessen the danger of infection of the middle ears through the eustachian tubes. If the bleeding recurs, the pack must be reinserted, preferably an iodoform pack which inhibits the growth of organisms and lessens the danger of otitis media.

Atelectasis is evidenced by fever, rapid respirations, cough, perhaps dyspnea and cyanosis. The physical signs are dullness on the affected side, with absent breath sounds. Fluoroscopy showing failure of the lung or lobe to aerate, with displacement of the mediastinum toward the collapsed side, together with elevation of the diaphragm on the collapsed side, confirm the diagnosis. The treatment is to move the patient and, if possible, have him sit in a chair, encourage coughing, have him lie on his good side, administer carbon dioxide to cause hyperventilation. If these measures fail, aspirate the occluding plug of mucus and blood bronchoscopically.

Pneumonia is evidenced by the same symptoms as atelectasis, but the breath sounds on the affected side are increased rather than absent, and fluoroscopy shows that the diaphragm is symmetrical, the mediastinum is in the midline, and both lungs aerate equally.

Lung Abscess is evidenced by fever, cough, and the expectoration of a mouthful of foul pus, usually a week or two after operation. The diagnosis is confirmed by roentgenography. There is no recorded instance where lung abscess followed tonsillectomy in the head-hyperextended position.

Technic of Tonsillectomy under Local Anesthesia.—*Anesthesia and Position of Patient*—Tonsillectomy under local anesthesia is done with the patient sitting upright in a chair, the surgeon sitting in front on a stool, with a headlamp for illumination. A sterile sheet is placed over the patient, and a basin in his lap which he holds with both hands (Fig. 158, A). If the patient gags excessively, the throat may be sprayed first with 2 per cent pontocaine. There

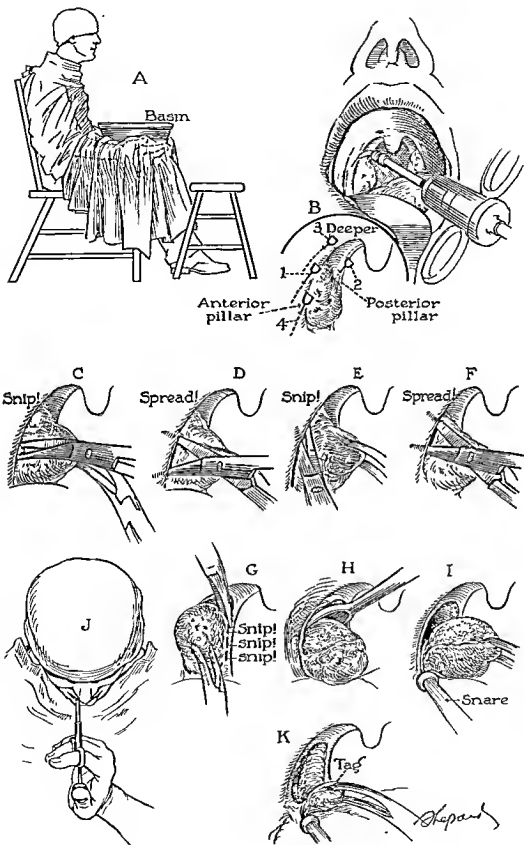


Fig. 158.—Technic of tonsillectomy under local anesthesia.

is evidence, however, that aspiration of blood and detritus with the danger of pulmonary complications is greatly increased by this procedure which anesthetizes not only the pharynx but to some extent the larynx.

Novocaine (1 per cent) containing a few drops of adrenalin to the ounce is now injected (1 or 2 cc) superficially into each anterior and posterior pillar (Fig 158, B), and the same amount deeply above the superior pole and into the lower pole. The plunger of the syringe should always be withdrawn slightly after introducing the needle and before injecting the novocaine to avoid accidental and sometimes fatal intravenous injection. After waiting a few minutes for the novocaine to take effect, the tonsil is grasped with the curved tenaculum. If there is any pain, 2 or 3 cc of novocaine may be injected directly behind the tonsil, by pulling the tonsil out and injecting deeply through the anterior pillar.

Removal of the Tonsils.—The tonsils are removed by scissors dissection and snare. The first snip is made with the scissors through the plica triangularis just behind the fold of the anterior pillar, the closed blades of the scissors then being spread widely (Fig 158, C). The incision is carried up around the upper pole and down the posterior pillar, spreading first with the scissors (Fig 158, D, E, F, G), then snipping as close to the margin of the tonsil as possible. A few bands of connective tissue between the upper pole and the tonsil fossa are cut with the scissors, the Hurd dissector is used to pull the tonsil down out of the upper half to two thirds of the fossa (Fig 158, H), and the remainder of the tonsil is taken off with the wire snare, slowly, so as to crush the blood vessels and favor rapid blood coagulation (Fig 158, I, J).

As soon as the tonsil is out, the patient inclines his head far forward over the basin, with the mouth open, so that any blood will run out and not down the throat. If the bleeding has not stopped after a few minutes, the fossa is gently sponged, the bleeding point is identified and grasped with a hemostat which is left in place for three or four minutes. Rarely will a suture be required. The tonsil fossa should be inspected by retracting the anterior pillar with the pillar retractor, and any remnants of tonsillar tissue grasped with the tenaculum and snared off. The lower pole especially must be searched for a lingual tag which can usually

be found and removed with the snare (Fig 158, K).

The scissors dissection technic, accomplished readily and often almost bloodlessly in the average tonsillectomy, is not as applicable where there have been previous peritonsillar abscesses with dense fibrous adhesions in the tonsil capsule. In these cases a sharp knife must be used, but since the demarcation between tonsillar tissue and pharyngeal muscle is obscured by the dense fibrosis, the proper line of cleavage is very difficult to discover. In these cases the snare will often do a better job of discovering the proper line of cleavage than the surgeon's knife, and the tonsil may be removed largely by the snare, piecemeal.

Postoperative Care.—While tonsillectomy under local anesthesia should be accomplished without pain, the effects of the novocaine soon wear off and the pain will be intense unless lessened by morphine— $\frac{1}{2}$ grain (10 mg)—given hypodermically as soon as the patient returns to bed and complains of pain. Thereafter codeine, $\frac{1}{2}$ grain (0.03 gm), and aspirin, 10 grains (0.6 gm) may be given every four hours as circumstances may require for pain, with nembutal—1.5 grains (0.1 gm)—and aspirin—10 grains (0.6 gm)—at bedtime for sleeplessness. An ice bag around the neck seems to lessen the pain somewhat and may lessen the tendency to hemorrhage. The patient is placed in the semi-reclining position for the first few hours to lessen venous congestion in the throat. Thereafter the postoperative care is the same as after the tonsillectomy with general anesthesia. Adults are advised to take a week off from work following tonsillectomy, though some are able to return to work sooner.

Recurrence of Tonsils and Adenoids.—The tonsils do not recur after a properly performed tonsillectomy. Superficial nodules of lymphoid tissue may develop in the tonsil fossa after any tonsillectomy, growing especially from the lingual tonsil, but these do not represent remnants of the original tonsil but rather a hypertrophy of the scattered follicles found throughout the mucosa of the pharynx. These superficial nodules of lymphoid tissue resting on the scar covering the tonsil fossa are easily differentiated from a true tonsil tag, which is always embedded beneath the scar covering the tonsil fossa. A true tonsil tag, because it is covered partly or completely by scar, which prevents normal drainage from its crypts, is a potential focus of

infection. The superficial lymphoid nodules, on the contrary, rarely cause trouble and rarely require removal.

While a completely removed tonsil never returns, the adenoids do not have a well-defined capsule permitting their complete enucleation, so that recurrence or regrowth of adenoid tissue is a frequent occurrence. This is particularly true in those instances in which the tonsils and adenoids were removed at an early age and in the resulting compensatory hypertrophy of the remaining lymphoid tissue the recurring adenoids may become large enough to cause symptoms and require removal.

GEORGE E. SHAMBAUGH, JR.

IRRADIATION TREATMENT OF HYPERPLASTIC LYMPHOID TISSUE IN THE NASOPHARYNX

Lymphoid tissue is normally an integral part of the mucous membrane of the pharynx and nasopharynx. It is so widely distributed—over the posterior part of the septum, the inferior wall of the sphenoid, and on the lateral and posterior walls of the nasopharynx and pharynx—that complete removal at operation is impossible. Recurrence in the nasopharynx after removal of tonsils and adenoids is so common as almost to be regarded as normal, especially in children. The extent of this hyperplasia varies enormously in both children and adults. The exact cause of this variation is not known, but we often see an excessive growth of lymphoid tissue in patients with infection, allergy, or ductless-gland disorders. Among the civilian population lymphoid hyperplasia is seen to a far greater degree in children than in adults, but in striking contrast to this are reports from more than one fighting front that in men of the various services is found an even greater amount of lymphoid hyperplasia than we are accustomed to see in children.

Infection in the upper air passages begins in lymphoid tissue. It is for this reason that we remove tonsils and adenoids. The failure of this operation always to prevent or markedly reduce the frequency and severity of recurrent infections is explained by the wide distribution of lymphoid tissue in this area, and that much

of it, dormant before operation, hypertrophies, producing the condition commonly known as "granular pharyngitis." This cycle of growth, often requiring repeated removal of adenoids, continues in many children until the age of puberty.

The nasopharynx is the most important location for a primary focus of infection in the upper air passages. This is attributable to the rich content of lymphoid tissue in its mucous membrane. Its proximity to the nasal passages, accessory nasal sinuses, and eustachian tubes accounts for many of the complications of the common cold.

Acute infections must be cared for with chemotherapy and surgical drainage, but it is also the duty of otolaryngologists to foresee and treat conditions in their patients which they know by experience may lead to frequent colds, cough, nasal obstruction, otitis and mastoiditis, sinusitis, asthmatic bronchitis, and other systemic disturbances. In addition to these complications due to infection, the condition of the nasopharynx plays an important part in the production of purely mechanical disorders of the ears and sinuses (aero-otitis or barotrauma) which are prevalent among aviators, submarine crews, and deep-sea divers, who are subjected to frequent and severe changes in temperature and barometric pressure. It is also well recognized that hyperplastic lymphoid tissue may be responsible for impaired hearing.

It is not the size, but the location of lymphoid nodules that is important. Even small nodules in the fossa of Rosenmüller, on the mesial wall of the pharyngeal orifice of the eustachian tubes, and in the area between the orifice of the tube and the posterior end of the middle turbinates, may interfere with the ventilating function of the eustachian tubes. These small nodules may be the seat of a low-grade chronic infection, or recurring acute infections that usually begin with a sore throat rather than nasal irritation and stuffiness. Since the region of the pharyngeal orifice of the tubes is rich in mucous glands, any infection of lymphoid tissue in this area causes an excessive secretion of thick mucus which further interferes with the function of the tubes.

Examination of Nasopharynx.—The condition of the pharynx and tonsils is easily and quickly determined, but examination of the nasopharynx is more difficult and often inaccurate if one depends solely on palpation or

inspection with a mirror. The nasopharyngoscope is just as essential for examination of the nasopharynx as the cystoscope is for examination of the bladder. This instrument is easily passed along the floor of the nose, even in children, and affords a clear view of every part of the nasopharynx. For several years I have employed the following technic for the examination of children and adults and have found it safe and time saving. First, the nasal passages are examined with a speculum for mechanical obstruction, such as deflected septum or polyps. Next all excess secretions are removed with suction and each side of the nose is lightly sprayed with 5 per cent butyl para-aminobenzoate. Butyl para aminobenzoate is as toxic as cocaine, but neither it nor cocaine is dangerous if used in amounts just sufficient to anesthetize the desired area, and if none of the solution flows backward into the pharynx and esophagus. Sufficient time must be allowed for the drug to act, and for this reason it is applied

tympanic catarrh) is loss of hearing for high tones. If school children are to have tests with the audiometer, the acuity for tones above 8000 double vibrations is most important. By testing for the lower tones and speech alone, the condition will not be recognized until the lesion is well advanced. If it is established that the child has high tone impairment, the tympanic membranes should be examined with an electric otoscope and the nasal passages and nasopharynx with the nasopharyngoscope. The changes commonly seen in the ear are retraction, especially of Shrapnell's membrane, opacity of the entire tympanic membrane, or hyperemia with fluid in the middle ear. The fact that the child's tonsils and adenoids have been removed, or that the tonsillar fossae and pharynx look normal, is no reason to assume that lymphoid tissue has not recurred in the nasopharynx. Most important of all is the necessity for doing something to correct conditions seen in the nasopharynx that interfere

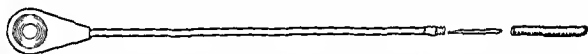


Fig. 159.—The applicator used to treat hyperplastic lymphoid tissue in the nasopharynx with radon. The radon is confined in the glass capillary tubing shown between the metal handle and the hollow brass container, which has an internal threading to fit that on the handle. The wall of the brass tube is 1 mm. thick. The length of the applicator is 6 inches. (From Crowe, S. J., and Burnam, C. F. Recognition, Treatment and Prevention of Hearing Impairment in Children, *Ann. Otol., Rhin. & Laryng.* 50: 15, 1941.)

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Considerations in Treatment.—When looking through a nasopharyngoscope the posterior end of the middle turbinates and the orifice of the eustachian tubes should be clearly seen. Normally the mucous membrane around the orifice of the tubes is perfectly smooth and pale pink in color. If the mucous membrane on the mesial lip of the eustachian orifices is red and granular or if lymphoid tissue fills the fossa of Rosenmüller and extends upward toward the posterior end of the middle turbinates, this constitutes a menace to the proper functioning of the eustachian tubes and predisposes the patient to barotrauma, frequent colds, and attacks of otitis media and sinusitis.

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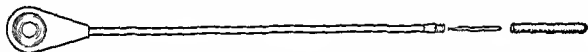


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If the adenoids are very large and no normal landmarks are seen in the nasopharynx, surgical removal, supplemented with irradiation, is preferable to irradiation alone, since it greatly reduces the mass of tissue and consequently the number of irradiation treatments. If the

adenoids are acutely infected irradiation like operation should be postponed for at least a month after the acute symptoms have subsided. If the nasopharynx is chronically infected the usual irradiation dosage of 2 gram minutes should be divided into two parts and given a week apart. This fractionating of the dosage avoids the acute exacerbation of infection that would invariably follow the administration of a full dose.

As a rule the best treatment for lymphoid tissue in the nasopharynx of children whose tonsils and adenoids have been removed is irradiation with a nasal applicator and not a second operation. The exception is when the adenoids are extremely large. Then the mass of lymphoid tissue in the midline should be removed and that remaining around the orifice of the tubes irradiated.



Fig. 160—Sketch of sectioned nose to show position of radium applicator during a treatment. The applicator is passed along the floor of the nose until it touches the posterior wall of the nasopharynx. Note that the applicator is near, not in, the orifice of the eustachian tube.

We do not advocate the indiscriminate use of radium as a treatment for deafness. It may or may not improve the hearing in a given case depending on the cause and duration of the impairment. Radium is of great value in reestablishing the lumen and the function of a eustachian tube partially occluded by hyperplastic lymphoid tissue. In other words it is used to correct a condition that predisposes to impaired hearing and to attacks of otitis media. The best results are obtained when it is used in children with beginning impairment of hearing. Only rarely are spectacular results obtained in extremely deaf children or in adults.

Treatment—Roentgen Rays—It is a comparatively simple matter to irradiate the nasopharynx with roentgen rays. The rays may be directed through the open mouth but this method of approach is difficult even in adults

owing to the time required for adequate treatment and the necessity for using a depressor to keep the tongue out of the field. For these reasons it is even more difficult in young children. If the rays are directed through the neck the principle of cross firing must be employed. Since approximately half of the rays delivered at the skin surface are absorbed by neck tissues before they reach the nasopharynx it is evident that several portals must be used in order to prevent skin irritation. The fact that a large percentage is absorbed necessitates a dosage on the skin surface much larger than is necessary to correct the lesion in the nasopharynx for which the treatment is given. This excessive dosage subjects the centers of ossification for the growing bones of the face and skull to a large amount of radiation which may do harm in a child and is of no value in the treatment of the primary condition in the nasopharynx. Furthermore it is difficult to maintain young children in a position for accurate cross firing for the duration of the treatment. Successful results in shrinking lymphoid tissue in and around the orifice of the eustachian tubes and in restoring the hearing cannot be expected unless the central beam in cross firing actually passes through the area where treatment is most needed. Finally roentgen ray treatments should not be carried out by anyone except an experienced radiologist.

Radon or Radium Application—In contrast to roentgen ray administration the radon or radium nasal applicator can be used by the otolaryngologist who tests the hearing, examines the nasopharynx and places the applicator on the exact spot where the treatment is most needed (Fig. 160). The applicator is small enough to pass along the floor of the nose of an infant. Only local anesthesia is necessary. With a strong radon applicator a three minute treatment is equivalent to fractional doses of roentgen ray that must be extended over a period of two or three weeks.

Next to the sex cells lymphocytes are the most sensitive cells in the body to beta and gamma irradiation. Therefore in treating patients the dosage employed is so small that there is no danger of a burn or a dry nasopharynx in which crusts form provided the rules concerning treating the nasopharynx during an acute or chronic infection are followed. The action of beta and gamma irradiation is to inhibit mitosis in the germinal centers and thus stop the

formation of new lymphocytes. Observation of hyperplastic lymphoid tissue under this treatment leads us to believe that lymphocytes, like skin cells, have a brief life cycle, probably not more than two weeks. Under irradiation treatment no new lymphocytes are formed to replace those discarded and the mass gradually shrinks and disappears, leaving the nasopharynx covered with smooth mucous membrane not unlike that on the nasal septum. If the dosage is just right the result is perfect and there is no recurrence. If the dosage is too great, ecchymoses appear in the nasopharyngeal mucous membrane, but there is no excuse for ever getting a burn.

At the Johns Hopkins Hospital we use from 800 to 1000 millicuries of radon with a brass filter 1 mm. in thickness (Fig. 159), but a more practical applicator for use in the Army and in civilian hospitals and office practice contains 50 mg. of anhydrous radium sulfate. The effectiveness of irradiation emitted through the applicator walls is exactly the same whether radon or a radium salt is employed. The dosage or the number of minutes the applicator is left in the nasopharynx depends on the amount of radium or radon in the applicator, the material used for screening, and the thickness of the walls of the radium containing chamber. The radium salt applicator should be made of monel metal. The handle is 25 cm. in length, and the radium containing chamber 15 mm. in length, 15 mm. in diameter, and 0.3 mm. in thickness. This allows the passage of more beta rays than the 1 mm. of brass in the radon applicator, but the results are as good as those obtained with the brass screening and the dosage is constant, six and six-tenths minutes on each side. After the radium salt is introduced into the applicator, the joint between the radium containing chamber and the handle should be braised in order to avoid any chance of the tip coming off in the nasopharynx or nose and the radium being swallowed. Braising the joint also prevents the escape of emanations or the seepage of moisture into the radium containing chamber. After the radium salt has been packed in the applicator it requires a month for equilibrium to be reached. Only then can the output be measured and the time required for safe nasopharyngeal treatments accurately determined. If, by any chance, even a microscopic hole appears in the thin wall of the radium-containing chamber, emanations will escape and render that appli-

cator unfit for treatment of patients. To guard against this the applicator should be tested weekly with a small alpha-ray electroscope. These are some of the precautions that everyone who treats patients with radium should practice.

In giving irradiation treatments the best protection for the operator is a distance of 30 feet from the applicator. Two pairs of thin rubber gloves will protect the hands from the beta rays while handling the applicator, but the use of a lead containing apron or gloves, as used in roentgen ray work, is of no value in protection against gamma rays. When not in use the applicator should be kept in a 7/16 inch hole in the center of a lead cylinder which is from 4 to 6 inches in diameter, depending upon the amount of radium or radon in the applicator. This hole contains a glass tube filled with alcohol. Heat must never be used to sterilize the applicator. Before inserting the applicator into the patient's nose, the excess alcohol should be washed off and the applicator dipped into hog-glycerin. This prevents nasal secretions from sticking to the applicator and is important, because the hands must never be used to wipe the applicator. The first evidence of too much exposure of the hands shows in the nails, which become ridged and cracked. It is also wise to have a complete blood examination at intervals, but with care there is no danger, as evidenced by the fact that many of us have given these treatments several times a week since 1928.

SAMUEL J. CROWE

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PHARYNGEAL PHASES OF BLOOD DYSCRASIAS

Hematology is an important science to the laryngologist, for in his field of endeavor many of the hematopoietic diseases have their initial manifestation. The mucous membrane of the nose and throat affords an easy and reliable field for the study of the manifestations of the hematopoietic diseases. There are primary blood diseases manifesting their initial lesion in the mouth and throat, likewise there are infections of the throat that influence the hematopoietic tissues to produce blood dyscrasias and at times it is very difficult to separate the borderline cases.

The older physiologist considered blood as a special form of connective tissue with a fluid matrix and having a twofold office (1) to carry to the tissue materials for their nourishment, and (2) to take away from the tissues the by-products of metabolism. Today, blood is considered a mixed secretion, whose formed elements are the results of parent cells living outside of the blood stream and whose functions include oxygen transportation by the red cells, osmotic pressure maintenance by the plasma proteins, ability to coagulate through the prothrombin-fibrinogen mechanism, nutrition with special reference to the role of the plasma proteins, and participation in body response to infection through complement and circulating antibodies and phagocytosis.

Hematopoiesis—In the adult the formation or production of blood normally occurs in the red bone marrow, the reticulo-endothelial system, and the lymphatic tissues. It is not known how only matured cells are released for evacuation into the blood stream. The action of this barrier is the unknown factor of hematopoiesis. Many theories have been advanced to explain the destruction of worn-out blood cells, but none of them have survived critical analysis. There are at all times sufficient immature blood

cells in the hematopoietic tissues that, if allowed to migrate into the blood stream, they would cause a dyscrasia.

Anemia—Anemia is a deficiency in the number of red blood cells (*oligocythemia*) or a reduction of the hemoglobin (*oligochromemia*) manifested clinically by a pallor of the skin and mucous membrane, palpitation of the heart, and a shortness of breath. We are born with a relatively high red cell and hemoglobin value. Pernicious anemia rarely occurs in children, it has a high incidence in persons of middle and old age. The normal menstrual cycle does not influence the percentage of erythrocytes or hemoglobin values. The old classification of primary and secondary types has been discarded. In its place, we have the following classification by Kracke¹:

Normal Decreased Increased	NUMBER	HEMOGLOBIN CONTENT		SIZE
	Normocythemic Hypocythemic Hypercythemic	Normochromic Hypochromic Hyperchromic		Normocytic Microcytic Macrocytic

Anemia of Acute Hemorrhage—This type of anemia is the result of loss of blood outside or within the body, manifested by a rapid feeble pulse, a lowering of the blood pressure, air hunger, and restlessness. The immediate hemogram is practically normal, the first change being an increase of the blood platelets as a means of decreasing the clotting time followed by a shift to the left due to the number of immature white cells. Not until the blood volume has improved by the transudation of body fluids into the circulation or the infusion of normal saline will the anemia be registered. This knowledge is practical in the differentiation between surgical shock and hemorrhage, for in shock there is no alteration in the cellular content, whereas, following the hemorrhage, the anemia becomes evident and increases as the blood volume is restored. An acute loss to 50 per cent demands immediate transfusions of whole blood and the avoidance of surgery until hemoglobin of 75 per cent has been reestablished.

Anemia of Acute Infections—This is the result of direct destruction within the blood stream and an inhibition of the hematopoietic tissues. Septicemia of *Streptococcus viridans* is usually malignant and accompanied by an increase of the white cells. Transfusion of whole blood corrects the anemia by replacement of the red cells and furnishes antigens to destroy the infection.

Anemia of Malignant Diseases—Anemia follows malignant disease of any tissue, but is profound when the hematopoietic tissues are involved, and at times the degree of anemia is out of proportion to the extent of the malignant process. The type is hypochromic, the reduction in hemoglobin content being greater than the reduction in the number of red blood cells with a resulting color index of below one. The cells are microcytic and may exhibit degrees of anisocytosis or poikilocytosis. The treatment is supportive and temporary.

Anemias of Vitamin C Deficiency—This anemia is a hypochromic type such as occurs in scurvy, in Osler's disease (nasal telangiectasia) following the epistaxis, or results from hemorrhage. The platelets are normal and likewise the bleeding and coagulating time, leaving as the cause brittle capillaries. Treatment consists in controlling the hemorrhage and the inclusion in the diet of citrus fruits or the administration of vitamin C.

Pernicious Anemia (Addisonian Anemia) (Biermer's Anemia)—This is a fatal disease unless properly treated, of unknown cause, characterized by a burning tongue, an absence of hydrochloric acid in the stomach, and periods of spontaneous remission and relapse. To explain this disease, it is necessary to consider that in the diet there is an extrinsic factor and that the glands of the stomach and upper duodenum secrete an intrinsic factor which is stored in the liver and released when needed. If there is a reduction of either of these factors the normal development of the erythrocytes will be prevented. The symptoms are usually those of the gastro intestinal tract. Glossodynia with a swollen beefy tongue is seen in most of the patients, the anterior half of the tongue being the worse.

The hemogram is characteristic when the patient is in relapse and the red cells fall to extremely low levels of 1,000,000 to 2,000,000 per cu mm with marked nuclear changes such as the formation of Cabot's ring and Howell Jolly bodies. Leukocytic changes are not characteristic, but the tendency is towards leukopenia. The platelets are correspondingly reduced.

Treatment consists in the administration of liver, hydrochloric acid, and vitamin B.

The Leukemias (Myelogenous, Lymphatic, and Monocytic)—The leukemic state is the result of a hyperplasia of the hematopoietic tissues and the type depends upon the tissues

involved. Piney² contends that all leukemias are neoplastic.

Lymphatic Leukemia—Lymphatic leukemia is either acute or chronic, usually fatal, and is characterized by hyperplasia of the lymphoid tissues manifested as glandular enlargements with an increase of lymphocytes in the blood.

CHRONIC LYMPHATIC LEUKEMIA—The etiological factor is unknown. The disease occurs most frequently in males past twenty years of age.

This leukemia is insidious and is often discovered during a general physical examination brought about by the presence of painless enlarged glands or the consciousness of increasing weakness with or without pallor because of the accompanying anemia. Vertigo and tinnitus may be the chief complaints although occasionally the patient complains of a dry and non-productive cough.

The general examination may reveal a patient of moderate pallor with the symptoms of anemias and an enlargement of the lymphatic glands. The anterior and posterior cervical glands are equally enlarged, discrete, and follow the normal pattern of the region involved. The spleen is enlarged and at times the liver is palpable below the costal margin.

The mucous membrane of the mouth and nose is pale in proportion to the anemia present. The tonsils are enlarged and of the active glandular type, but the adenoid tissue does not regenerate or hypertrophy. During the late stages the lymphatic elements of the gums hypertrophy to enclose the teeth. As in pyralism, this phenomenon does not occur in the gums after the complete removal of the teeth. (I once examined a woman with chronic lymphatic leukemia whose upper jaw was edentulous and remained void of lesions, while the lower gingival process containing teeth manifested a lymphatic hyperplasia sufficient to hide the teeth.) The sinuses are not involved, nor are the pneumatic spaces of the mastoids. Visual disturbances are the result of retinal hemorrhages. The cranial nerves, especially the seventh and eighth, are frequently silenced by tumor infiltration.

The leukocytic count ranges from 50,000 to 1,000,000 cells per cu mm of blood, the predominating cells being the small lymphocytes with prominent nuclei. As the disease progresses degenerated forms of lymphocytes appear. The erythrocytes and hemoglobin are

proportionally reduced with a color index below one. The platelets are moderately reduced.

The pathologic manifestations consist in lymphoid hyperplasia with an increase of lymphocytes in the blood and their deposit throughout the tissues of the body, especially in the lymphatic glands.

Generalized lymphadenopathy associated with the above mentioned blood picture is diagnostic. The disease must be differentiated from Hodgkin's, infectious mononucleosis, secondary inflammatory glands, and the glandular enlargements of syphilis and tuberculosis.

Röntgen ray therapy renders temporary improvement. Tonics containing arsenic are beneficial and because of the spontaneous remissions many remedies have enjoyed unearned credit. Transfusions are indicated according to the anemia.

The prognosis is poor and the disease is eventually fatal, although some of the patients live five or six years depending upon the activity of the tissues.

Acute Leukemia (Acute Leukosis)—Acute leukemia is a rapidly fatal disease whose initial appearance may be oral. The type depends upon the predominating cell, which may be myeloblastic, lymphatic, or monocytic. The disease occurs most frequently in children and young adults. It apparently is the result of an infection although no constant organism can be isolated.

The onset is sudden and resembles an acute tonsillitis accompanied by multiple lesions within the mouth. The course is febrile and the individual becomes increasingly ill. Severe epistaxis is common and, in time, hemorrhage occurs from all the mucous membranes.

Upon examination the patient appears acutely ill with visible enlargement of the lymphatic glands. The spleen is palpable and the enlarged intra-abdominal glands are tender. The lesions of the mouth are multiple, beginning as a hypertrophy of the pre-existing lymphatic structures and terminating in necrosis. The involved tissues bleed readily and the hemorrhage at times is profuse. The cranial nerves are less likely to be injured than in the chronic type of lymphatic leukemia. The mastoid process may be the site of an acute lymphatic deposit presenting the characteristics of an acute suppurative mastoiditis.

The anemia progresses with the disease and is severe following the hemorrhages. The number of blood platelets is reduced and does not

increase after the hemorrhages. The type of immature leukocytes determines the leukemic state. In the beginning the total white blood cell count is reduced (aleukemic state) to 3000 or 4000 per cu mm while the terminal count may be over 100,000.

When a youthful ill patient presents multiple lesions of the mouth and throat, the blood should be carefully studied. Before the total count has alarmingly increased, there should be sufficient immature leukocytes to warrant a diagnosis of acute leukemia. The late diagnosis is definite—a high white cell count with predominance of immature cells (the prognosis becomes increasingly grave when the blast forms predominate), a marked reduction of platelets, and an anemia. Although the condition is rare, it is seldom confused with other acute diseases such as diphtheria, acute streptococcus stomatitis, Vincent's disease, or secondary syphilis.

The treatment is symptomatic and palliative. The mouth is cleansed with a thin paste of alcaïroid powder. Röntgen ray therapy is useless and multiple transfusions are futile.

Chronic Myelogenous Leukemia—This disease is of interest as the initial symptom may be hemorrhage as an epistaxis or retinal with a sudden loss of vision.

The findings on examinations are similar to those of chronic lymphatic leukemia. The anemia follows the course of the disease, although at times it resembles pernicious anemia. The leukocytes increase to as high as 1,000,000 per cu mm. The shift is to the left. The neutrophilic myelocyte count becomes 50 per cent of the total white count.

The diagnosis is arrived at when the blood picture just described is encountered in a middle-aged individual with an enlarged spleen. When the leukocytic count is moderately high, the disease must be differentiated from an acute infection associated with a high white cell count.

Röntgen ray therapy is satisfactory and should be augmented by the administration of arsenic.

The total count is not as important as the percentage of immature cells, when arriving at a prognosis. Many of these patients have spontaneous remission of good health and live three or four years after the diagnosis has been made.

Infectious Mononucleosis (Benign Lymphadenosis)—Pfeiffer³ established the existence of the disease as a clinical entity and gave it the

name of "glandular fever" in 1889. Subsequent observers agree that the disease is infectious and numerous epidemics have been reported. The frequency of outbreaks in our universities, colleges, and high schools of an acute infection of the throat, complicated by glandular enlargements, has attracted attention to infectious mononucleosis. Sporadic cases in adults occur, but are missed, because of the failure to have routine blood studies made in office practice.

The etiologic agent is a virus, as considered by Barber,⁴ whose three patients had ulcerations of the tonsils, resistant to treatment. Sporadic cases usually occur in adults, while epidemics occur among children. The incubation period is from five to fifteen days. There are two views regarding the cause of the response of the blood: (1) that a specific infective agent gives rise to the characteristic lymphocytic response, and (2) that the lymphatic reaction is probably due to an unusual constitutional disposition on the part of the patient reacting to an ordinary acute infection.

The onset is gradual, the patient complaining of fatigue, dull headache, and general malaise followed by fever, sore throat, and lymphadenopathy of the cervical, axillary, and inguinal regions.

The physical examination reveals an angina with an enlargement of the tonsils and cervical lymph glands, an elevation of temperature, and an enlargement of the spleen. Suppuration seldom occurs, unless complicated by a mixed infection. The posterior cervical lymphatic chain enlarges and sometimes the deeper chain under the sternocleidomastoid muscle.

The clinical importance of a diagnosis is the mental relief afforded—that the individual has a reason for the protracted disease which appeared at first to be a simple tonsillitis and that a more serious condition, such as acute lymphatic leukemia or Hodgkin's disease, is excluded.

The red blood cells and the hemoglobin remain normal or show a late secondary anemia. The total white count is not typical. If made early, it will reveal a leukopenia from 3000 to 4000 cells per cu. mm., and as the glands enlarge it increases to 20,000 or more, 50,000 cells suggest an acute leukemia. In children, the leukocyte count may reach its maximum at the end of the first week, whereas in an adult more time is required. The hemogram displays a shift towards an increase in mononuclear cells

at the expense of the polymorphonuclear neutrophils. The mononuclear cells include both monocytes and lymphocytes. The former, in normal numbers, are slightly increased, while the lymphocytes show a relative and absolute increase of 50 per cent or more. The abnormal blood picture may persist for months after the initial attack.

Headache is a common symptom in adults, although the number of cells in the spinal fluid will average less than 100 per cu. mm.

The disease may be confused with diphtheria, mumps, follicular tonsillitis, streptococcal pharyngitis, or sinusitis, accompanied by cervical adenitis. The septic cases must be differentiated from pyogenic septicemia, acute leukemia, acute Hodgkin's disease, and the acute fevers characterized by lymphocytic hemic responses.

Kaufman⁵ contends that 2 to 10 per cent of infectious mononucleosis patients will give a false Wassermann reaction and suggests that in laboratory procedures, false positive reactions be checked by the heterophil antibody test. The cause of the false positive reaction in infectious mononucleosis is unknown. It usually appears during the second week and may persist for several months and affects flocculation procedures, such as the Kahn test and the Wassermann reaction. The heterophil antibody test is diagnostic when positive in the higher titers. Agglutination in any dilution above 1:64 is considered diagnostic if there is no history of serum sickness. The titer seems to be higher when the temperature is higher and remains elevated for months.

The symptomatic treatment of the anginous type of infectious mononucleosis may be either refrigeration of the cervical glands by the use of cold compresses or the application of continuous heat, e.g., antiphlogistine packs and the daily use of radiation by the water-cooled ultraviolet will remove the tenderness and some of the swelling. Many clinicians advocate deep roentgen-ray therapy when the former fails or the glands involved belong to the deeper chain. Transfusions of convalescent serum assist in building up an immunity to the offending virus. Saltzman⁶ advises the intramuscular injection of 0.03 mg. of bismuth and potassium tartrate. Whether this pharmacologic compound has a specific action or its effect is due to increasing the number of polymorphonuclear neutrophils will require confirmation. Maintenance of thorough dental hygiene when administering the

bismuth therapy has been found to be a good practice, as the metal possesses a tendency to deposit itself about the teeth

Thomsen⁷ reported gratifying results in the treatment of infectious mononucleosis with sulfanilamide and convalescent serum. He contends that sulfanilamide alone exerts its beneficial effects upon the secondary invading organism.

The prognosis is favorable, complications are rare, and fatalities depend upon secondary infections.

Agranulocytosis (Malignant Neutropenia)—Although Gubler,⁸ in 1857, and Trousseau,⁹ in 1865, described an angina similar to agranulocytosis, credit must be given to Schultz¹⁰ for the term 'agranulocytosis'. Bethell advises the study of Plum's¹¹ work on agranulocytosis as the most authoritative and comprehensive discussion available.

Numerous observers agreed with Lovett¹² that the *Bacillus pyocyaneus*, because of its ability to produce leukopenia, was the offending bacteria, later the frequency with which the spirochetes of Vincent's and their associates, the fusiform bacilli, were isolated from the mouths of patients suffering from agranulocytosis brought these organisms under suspicion. There is no doubt that some cases are the result of severe sepsis, but no organism has consistently been associated with the disease.

Jackson¹³ personally collected 109 instances of the condition following the administration of drugs: sulfanilamide 34, sulfapyridine 8, aminopyrine 39, allonal 7, causalin 4, and stated that cibalgin, acetanilid, amidopben, cinchophen, amyral compound, bismarsen and neocinchophen also might incite agranulocytosis. Thompson¹⁴ reported a fatal case following sulfathiazole. Curry¹⁵ reported an acute agranulocytosis occurring after sulfadiazine therapy. Leser¹⁶ cites the work of Peper, Long, and Bliss, who noted that no instance of acute agranulocytosis had followed sulfathiazole, but Kennedy and Findland¹⁷ reported a fatal case following its administration. Acute hemolytic anemia may develop in the first twenty-four hours of administration of the drug, but it requires two weeks' administration before agranulocytosis develops. Pippin¹⁸ emphasizes the ominous importance of fever following the use of sulfathiazole. Kracke¹⁹ observed that eight out of nine patients seen by him with acute agranulocytosis had received coal tar products and suggested

that the benzene ring was of etiologic importance. Arsphenamine, bismuth, gold, and benzene when improperly used are capable of producing a neutropenia.

The ulcerations in agranulocytosis occur most frequently in the mouth, about the anus, or in the vagina—in areas where bacteria are constantly present. The lesion is a round-cell infiltration and is characterized by the absence of polymorphonuclear neutrophils. The exciting cause may be bacterial or chemical, but in either case it is the myeloid elements of the bone marrow that are affected. The severe reduction of the neutrophils is the result of their destruction in the peripheral blood stream or their migration into the viscera or the failure of maturation of the myeloid elements in the bone marrow. There is an unknown X factor in hemopoiesis which is a barrier that prevents immature cells from being dumped into the peripheral circulation. Perhaps in this disease the barrier prevents the normal discharge of mature granulocytes. There is little evidence to support the destruction theory and the idea of abnormal distribution lacks verification as the blood obtained from the organs varies slightly from that of the peripheral circulation. Apparently, then, it is failure of maturation which is responsible. Occasionally the shift occurs before a lesion can be isolated. Jackson and Parker²⁰ reported a case in which this occurred five days before the sore throat was visible. The author observed a fatal case for three days before the appearance of a lesion, in which the blood picture was typical of agranulocytosis.

The local care of the ulcerations should be limited to mild oxidizing agents and the avoidance of caustics. At times the lesion about the tonsil will imitate an early peritonsillar abscess. Surgery should be avoided, and even aspiration is dangerous. Administration of vitamins is of temporary advantage, especially nicotinamide. Transfusions are temporary measures and, since the blood is expendable, must be repeated according to the demands. Watkins²¹ advocated the administration of yellow bone marrow concentrate for several days. Pentnucleotide (40 cc a day) is the most potent drug available. Conservative application of roentgen-ray therapy was recommended by Dowdy and Lawrence²² by Murphy,²³ and by Lawrence²⁴.

Leser²⁵ advises intensive parenteral liver therapy with repeated blood transfusions and 10 cc of pentnucleotide administered four times a

day Davies and Wingfield²⁶ utilized injections of epinephrine to increase the polymorpho-nuclears when pentnucleotide therapy and blood transfusions failed

Special Considerations in Treatment of Blood Dyscrasias—Transfusion—When possible, whole blood should be administered to replace blood that has been lost. The employment of whole blood is rarely contraindicated, one of the exceptions being in extreme hemoco-concentration, such as may occur following severe burns. Plasma and serum have an advantage in emergencies because compatibility tests are not necessary. Preliminary observations indicate that purified human albumin is a safe and effective substitute for plasma. The problem of providing blood substitutes in large quantities will be greatly simplified if nonantigenic fractions of animal plasma proteins can be prepared. The administration of plasma has the advantage of being moderately safe regardless of the blood group, because iso-antibodies will be diluted sufficiently to prevent agglutination.

Levine and State²⁷ call attention to cutaneous tests for the presence in plasma of A and B factors, these being the same antigens as those occurring in the erythrocytes of A and B blood. This sensitivity is not specific to any special blood group and Levine and State's experiments conclusively prove that there are no reactions following the intravenous administration of plasma, which gives a negative cutaneous test. These investigators advise cutaneous testing as a valuable adjunct to plasma therapy.

In spite of blood grouping and cross matching, reactions follow the administration of whole or citrated blood. In the beginning this was blamed upon the A and B agglutinogens, but Landsteiner and Wiener²⁸ described a new factor called by them the "Rh agglutinin." This factor was prepared by immunizing rabbits against rhesus monkey blood and is present in about 85 per cent of individuals tested. If members of this 15 per cent Rh negative group have repeated transfusions of blood from Rh positive patients, they will produce an antibody capable in future transfusions of causing anaphylaxis. This condition is exaggerated in post-partum transfusions if the wife is a Rh negative individual and the husband as donor is Rh positive. Most likely the offspring would be Rh positive and the mother, owing to her contact

with the fetus, would have developed anti-Rh bodies. In this situation there is a tendency in some women to bear offspring all of whom except the first develop erythroblastosis.

Vitamin K Therapy in Re-establishment of Normal Clotting Time—According to Howell's theory of coagulation,²⁹ adequate ionized calcium, thromboplastin, and fibrinogen must be present for the normal clotting of blood. When the thromboplastin is liberated by tissue injury in the presence of vitamin K, it is converted into prothrombin, which when combined with calcium forms thrombin, a fibrin ferment capable of converting the fibrinogen into fibrin. Vitamin K₁ is a product of alfalfa and K₂ is obtained from putrefied fish meal. It is accepted that prothrombin is a product of liver activity and that the absorption of vitamin K is favored by the presence of bile salts. Synthetical vitamin K substances administered orally are capable of re-establishing normal clotting time, when the deficiency is due to a lack of prothrombin.

The Sulfonamides in the Control of Vascular Oozing—The use of one of the sulfonamides as a dusting powder in operative wounds has been recommended for the control of moderate oozing. Cunningham³⁰ states "the application of powdered sulfapyridine and sulfamethylthiazol to experimental wounds in the horse, guinea pig and rabbit results uniformly in the control of vascular oozing. These results confirm observations made in cases of delayed and secondary tonsillar hemorrhage in which the aforementioned drugs were used successfully in the control of persistent bleeding."

In our experiences this happy promise of aid in the controlling of persistent vascular oozing of secondary tonsillar bleeding has not been fulfilled.

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NEOPLASMS OF THE PHARYNX AND FAUCES

Benign Neoplasms of the Pharynx and Fauces.—Benign neoplasms occur with comparative rarity in the pharynx Those most frequently found include papillomas, adenomas, fibromas, lipomas, hemangiomas, and cysts

Types of Lesions—*Papillomas* are probably the commonest type of benign tumor found in the pharynx They may grow from any part of the mucous membrane, but most often arise from the base of the uvula and from the anterior pillars As a rule, they are pedunculated but occasionally may be sessile They should be removed surgically or destroyed by electrocoagulation

Adenomas are occasionally found in the region of the uvula and soft palate, rarely on the tonsils They usually grow just under the mucous membrane and are firm in consistency If they should become too large or produce disturbing symptoms, surgical removal is indicated

Fibromas are usually located on the posterior pharyngeal wall. They present a smooth rounded appearance and are covered by normal mucous membrane This type of tumor varies considerably in size and the treatment depends entirely on the anatomic location and the size of the growth. By means of suspension laryngoscopy it is possible to remove fibromas from the pharynx and hypopharynx provided they are not unduly large In the latter case it may be necessary to approach the growth through an external incision

Hemangiomas of the posterior pharyngeal wall are occasionally seen as bluish tumors of bizarre shape and size They may be sessile or pedunculated and, if large, they bleed easily Radon seeds or radium needles embedded in the mass will reduce the size and electrocoagulation may be used to destroy the growth.

Cysts in the pharynx frequently involve the valleculae on the lingual surface of the epiglottis and at times reach such large dimensions as to interfere with deglutition and respiration They are best exposed by direct laryngoscopy and must be completely removed so as to preclude the possibility of recurrence

Other benign tumors occasionally harbored in the pharynx include lipomas, myxomas, chondromas, neurofibromas, lymphomas, and tera-

tomas Mixed tumors also may occur in the pharynx. They always present a smooth, firm, hard mass on palpation.

Surgical Treatment—Benign tumors should always be removed completely with as little trauma to the normal mucosa as possible. Tumors of the hypopharynx are best removed by utilizing suspension laryngoscopy, which permits the use of both hands and makes possible the removal of tumors and also the control of bleeding by ligation of the vessels as far down as the mouth of the esophagus.

Malignant Neoplasms of the Pharynx and Fauces—Malignant tumors are more commonly found in the pharynx than benign tumors and usually appear after the age of forty. The incidence of squamous cell carcinomas is greater than that of sarcomas. Adenocarcinomas are occasionally encountered and recently a number of epidermoid carcinomas have been reported. Such growths may involve any portion of the pharynx, they may appear on the uvula, soft palate, faucial or lingual tonsils, or on the posterior and lateral walls of the pharynx. However, the site of predilection appears to be the lower lateral pharyngeal wall.

Malignant Tumors of the Tonsils—Malignant growths occurring in the tonsils may have their origin in any part of this structure. The lesion is not always characteristic but usually starts as a unilateral enlargement and thickening of one tonsil associated with mild hyperemia and discomfort. Malignant lesions occurring in the tonsils may be sarcomas, lymphosarcomas, or carcinomas. Clinically, it is practically impossible to differentiate one from the other. This can be done only by microscopic examination and is not of too great significance as long as the malignancy is recognized. Too often such lesions are treated as chronic inflammatory tissue or chronic ulcerations. The seriousness of delay in instituting proper treatment in such cases is obvious. In cases of unilateral enlargement of a tonsil which is inflamed and does not respond to local treatment within a few days, biopsy should be done. Thus, an early diagnosis of malignancy can be established and proper therapy instituted.

SIGNS AND SYMPTOMS—The earliest definite evidence of unilateral enlargement of the tonsil is the presence of an ulcer with a hard and irregular edge. Growth is rapid and the adjacent tissues show early invasion. The information gained by palpation relative to the extent of

induration is most valuable in determining the course of treatment. In the very early cases the cervical glands may not be involved, whereas in the more advanced cases the glands at the angle of the jaw may be palpable or even matted together. The symptoms are in direct proportion to the extension of the lesion. If the pillars, tongue, and posterior pharyngeal wall are involved, dysphagia is present, movements of the tongue are restricted, and there is always pain radiating to the ear. As the growth progresses there is a feeling of fulness, more pain, excessive mucus, and occasionally some bleeding.

PROGNOSIS—Successful treatment of malignant disease of the tonsil depends largely on the extent of the lesion. In those cases in which the lesion is confined to the tonsil only, the prognosis is good if adequate treatment is instituted early. However, if the lesion has extended to the adjacent tissues or the glands show involvement, a guarded prognosis must be given.

TREATMENT—In the extremely early stage when the lesion is confined only to the tonsillar tissue, complete enucleation of the tonsil followed by radiation should give excellent results. In more advanced cases, roentgen ray therapy is advocated and surprisingly good results are frequently obtained. If the lesion has invaded the adjacent tissue and glands, the results from radiation will not be good. In these latter cases, surgical measures are contraindicated.

Pharyngeal Carcinoma and Sarcoma—Carcinoma of the pharynx occurs most frequently on the lower lateral walls, although the posterior wall is sometimes involved. The epidermoid carcinomas are more common than the adenocarcinomas. Lesions on the posterior pharyngeal wall at first involve only the mucosa of the pharynx and gradually invade the deeper structures. Such a lesion first appears as a firm, elevated growth which quickly breaks down in the center, leaving an ulcerated, granular surface covered with mucopus and surrounded by a crater-like rim well defined with elevated and indurated margins. In the early stages there is only vague discomfort such as a sensation of fullness and slowly progressive pain. Extension is rapid and the adjacent tissue as well as glands show early invasion. Because such lesions are easily seen, an early diagnosis should be expected. If the lower lateral pharyngeal wall is

involved, the growth is usually fairly extensive. Thus, pain and discomfort are late manifestations of a malignant growth in this region. There is usually an indurated nodular mass frequently extending into the pyriform sinus. With the appearance of ulceration pain in creases and dysphagia develops. Cervical glands show early involvement and pain radiates to the ear on the same side. Mirror examination permits, in most cases, visualization of the lesion, or may reveal a fullness or stagnation of secretion in the pyriform fossa which is suggestive of an existing pathologic condition. The lower lateral wall, pyriform fossas, and mouth of the esophagus should be examined directly and a specimen for biopsy obtained.

Sarcoma of the pharynx may develop on the posterior or lateral pharyngeal walls. The symptoms and appearance of sarcoma are identical with those of carcinoma and differentiation can only be made microscopically. Lymphosarcoma is the type of tumor usually found.

PROGNOSIS—Early diagnosis is imperative if a cure is to be obtained. The gradation of tumors will help to determine the radiosensitivity as well as the relative malignancy of the growth. Early lesions involving the posterior pharyngeal wall offer a better prognosis than those growths occurring on the lateral pharyngeal walls and pyriform fossas. The prognosis of malignant growths of the pharynx is bad and the presence of indurated glands serves only to increase the gravity of the prognosis.

TREATMENT—Splendid results can be obtained by surgical extirpation if malignant pharyngeal lesions are recognized early. However, few cases are seen early enough to be cured by surgical measures. As a rule, surgical removal is contraindicated in the presence of extensive malignant tumors. However, growths on the lateral pharyngeal walls may occasionally be successfully eradicated by means of lateral pharyngotomy.

It is a generally accepted fact that irradiation is the method of choice in the treatment of malignant lesions of the pharynx. It should be remembered that localized differentiated lesions respond better to irradiation than the undifferentiated ones. Adequate radiation therapy should be instituted, as excellent results have been obtained in some cases previously considered hopeless.

FRANCIS E. LEJEUNE

SUSPENSION LARYNGOSCOPY

As a surgical approach to lesions of the hypopharynx and base of the tongue, suspension laryngoscopy should be given serious consideration. The suspension laryngoscope, originally conceived by Gustave Killian and perfected by Clyde Lynch, is primarily intended for the direct examination of the larynx and as a means of approach in intralaryngeal surgery. It may also be used advantageously in cases of supraglottic tumors and of new growths occurring in the hypopharynx, base of the tongue, and mouth of the esophagus. This method of direct visualization of the larynx and hypopharynx presents a distinct advantage in that both hands are free to be used in any surgical procedure in this area. The ability to retract or palpate a lesion occurring in the hypopharynx or larynx is of considerable clinical significance. Dissection of tumors from the hypopharynx and the control of hemostasis by sutures can easily be accomplished if suspension laryngoscopy has been employed. Tumors of surprisingly large size may be removed with little or no difficulty and electrocoagulation is successfully carried out by this method. In addition, the suspension laryngoscope is a valuable aid in removing certain types of impacted or embedded foreign bodies in the hypopharynx, larynx, and mouth of the esophagus. Although suspension laryngoscopy offers certain advantages, it is not the answer to all of the problems encountered in the treatment of lesions of the hypopharynx and larynx. For this reason, it should be used in conjunction with other recognized methods of direct laryngoscopy.

Suspension laryngoscopy can be carried out on any patient whose mouth can be opened sufficiently to introduce the tongue spatula and whose neck permits extension. Patients with receding lower jaws are not good subjects for suspension laryngoscopy. Invariably a poor or inadequate exposure of the hypopharynx or larynx is obtained.

The Instrument—The suspension laryngoscope comprises two distinct parts. The laryngoscope proper consists of a mouth gag and tongue spatula combined and attached to a shaft which serves as the body of the laryngoscope, which has at its upper end a hook for attachment to the crane. The shaft of the laryngoscope is sturdy. The mouth gag is formed by

a pear shaped ring and has on its lower border a place for the attachment of the tooth plates which when in position fit against the upper teeth. It is always advisable to protect the teeth and for this purpose a piece of sheet lead molded to the shape of the patient's teeth serves to protect the enamel from possible in-

jury. The mouth gag is opened and closed by means of a thumb screw and may be opened to one and three fourths inches. There are six different sized tongue spatulas intended for use on patients ranging in size from infants to adults. A wing screw near the lower end of the shaft serves to elongate the tongue spatula from one-fourth to one-half inch. Near the middle of the

shaft there is a joint known as the worm gear joint which serves to flex or extend the head (Fig 161 B). The second part of the laryngoscope is a movable crane attached to the edge of the operating table near the head (Fig 161 C). Its adaptability lies in the fact that by means of

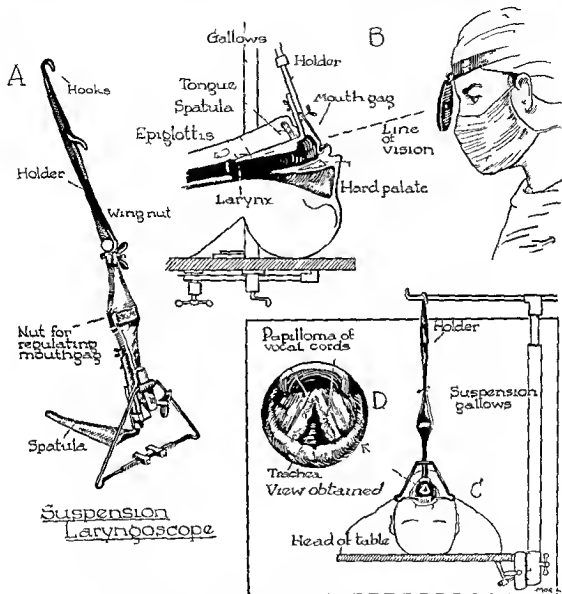


Fig 161 —A Suspension laryngoscope B Application of suspension laryngoscopy C, Front view of suspension laryngoscopy D Close-up view of larynx

jury. The mouth gag is opened and closed by means of a thumb screw and may be opened to one and three fourths inches. There are six different sized tongue spatulas intended for use on patients ranging in size from infants to adults. A wing screw near the lower end of the shaft serves to elongate the tongue spatula from one-fourth to one-half inch. Near the middle of the

two small cranks it may be used either as a horizontal or vertical crane.

Preoperative Preparation.—One hour before operation the patient is given $1\frac{1}{2}$ grains (0.12 gm) of secenal and forty five minutes before operation $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.01 gm to 0.015 gm) of morphine and 1/150 grain (0.0004 gm) of atropine are given.

Anesthesia—Suspension laryngoscopy may be satisfactorily employed under a local anesthetic but is more easily performed under a general anesthetic. For those patients requiring electrocoagulation, sodium ethyl (methyl butyl) thiobarbiturate (pentothal sodium) is the anesthetic of choice.

Technic.—Before the suspension laryngoscope is introduced, two sterile towels are placed under the patient's head. The lower one serves to cover the table while the upper one is wrapped around the head of the patient, covering the eyes which are protected by a piece of wet cotton placed over them. Complete relaxation, particularly in adults, is necessary. A side mouth gag is inserted and the suspension laryngoscope is introduced along the base of the tongue, care being taken to avoid any trauma to the posterior pharyngeal wall. The laryngeal surface of the epiglottis is engaged with the tip of the tongue spatula. If the selection of the spatula has been accurate, the tooth plates of the suspension unit will fit snugly against the lead plate previously attached to the upper front teeth. This plate is made of sheet lead and serves to protect bad dentures as well as the enamel surface of the teeth. The lead is easily molded to the shape of the individual teeth and helps to distribute the pressure which is exerted against the upper jaw. Slight traction is made on the suspension unit while the tongue is kept in the median line, at the same time by means of the thumbscrew the mouth gag is spread open and the tongue fixed. After the laryngoscope has been introduced into the mouth and the structures exposed it is then ready to be attached to the horizontal bar of the crane by means of the hooks on the end of the suspension shaft. The weight of the head on the table is taken up partly by the vertical crane and if the horizontal crane is brought forward it produces an upward tilting of the end of the tongue spatula to provide better exposure of the anterior commissure. Actually, the head of the patient is seldom if ever lifted completely off the table. Just enough upward pull is exerted by the crane to produce extension of the head sufficient to convert the curved tract from the teeth to the vocal cords into a straight line with the mouth wide open (Fig. 161, A).

The greatest difficulty in the performance of suspension laryngoscopy is the maintenance of the tongue in the median line and the exposure of the anterior commissure of the larynx. Fail-

ure to do either one of these would probably result in poor exposure of the larynx and possible trauma to the glossopalatinus muscle. Supraglottic tumors or lesions on the base of the tongue, lateral pharyngeal wall, or pyriform fossa may require insertion of the spatula to one side of the tongue for a better approach to the field. Exposure of the anterior commissure depends greatly upon the anatomic structure in this region. A long thin neck with poor muscular development is the ideal for suspension demonstration, whereas a short thick neck invariably presents difficulty in exposing the anterior commissure. By turning the handle of the crane which brings the crossbar toward the operator, it is possible to angulate the tip of the spatula upwards, thereby providing one eighth to one fourth inch more space, which at times is sufficient to visualize the anterior commissure.

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TECHNIC OF LATERAL TRANSTHYROID PHARYNGOTOMY FOR MALIGNANT DISEASE

Types of Growth—Before describing the technic of lateral transthyroid pharyngotomy, a few remarks regarding the general characteristics of malignant disease of the pharynx will not be amiss. Limiting ourselves to the laryngopharynx we have two groups, first, the epilaryngeal comprising growths of the (1) epiglottis, (2) aryepiglottic fold, (3) lateral wall of the pharynx, and (4) the pyriform sinus, secondly, the hypopharyngeal group, the growths of the postcricoid region. The operation of lateral transthyroid pharyngotomy is not necessarily limited to malignant conditions of the laryngopharynx. This method gives adequate access to this area and permits satisfactory treatment of a large number of growths both benign and malignant. Carcinoma in this area does not progress rapidly and we have, after the first symptoms arise, a period of a few months during which time

the case is still in the operable stage. Growths arising on the epiglottis, aryepiglottic fold, and lateral wall of the pharynx lend themselves to satisfactory operative procedure. Those arising

we have strong reason to suspect a postcricoid carcinoma. By means of lateral transthyroid pharyngotomy this entire area may be palpated through the laryngopharyngeal aponeurosis and



Fig 162



Fig 163



Fig 164

Fig 162 — Photograph of patient following removal of larynx and cervical esophagus and before plastic closure

Fig 163 — Photograph of patient following reconstruction of cervical esophagus

Fig 164 — Photograph of patient nine years following lateral transthyroid pharyngotomy for malignant growth of lateral wall of the pharynx

in the pyriform sinus which in the early stages are symptomless until the lateral wall of the larynx and thyroid cartilage are involved become almost inoperable except by extensive

operation can be definitely decided upon without opening the pharynx.

The one method of operative treatment in carcinoma of the laryngopharynx is the opera-



Fig 165



Fig 166



Fig 167

Fig 165 — Photograph of patient five years following removal of epiglottis and portion of base of tongue by lateral transthyroid pharyngotomy and bilateral block dissection of neck

Fig 166 — Photograph of patient after reconstruction of esophagus following lateral transthyroid pharyngotomy notice that the tracheotomy tube is completely clogged

Fig 167 — Shows base of tongue and growth removed with part of the lateral wall of the pharynx by lateral transthyroid pharyngotomy

operation. In the hypopharyngeal group occurring mostly in women, with early symptoms of obstructive dysphagia, frequent choking attacks during meals and huskiness of voice,

tion of lateral transthyroid pharyngotomy, this being the foundation on which all procedures, from the simplest to the most elaborate with plastic closures may be built up. The essential

facts are (1) It is a method of access (2) The wall of the pharyngeal aponeurosis is exposed

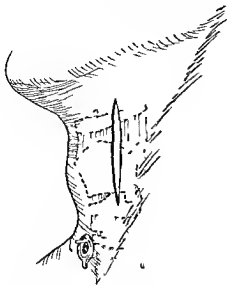


Fig 168—Shows line of incision for the simplest lateral transthyroid operation. The outline of the hyoid bone and alae of thyroid cartilage is shown graphically

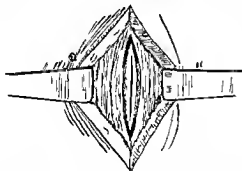


Fig 169—The infrahyoid muscles are split in line of incision down to and including external perichondrium.

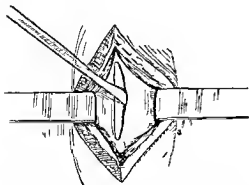


Fig 170—Elevation of external perichondrium from thyroid alae, this separation is continued on the under surface of thyroid wing

unopened by removing the great cornua of hyoid bone and ala of thyroid cartilage (3)

Through the exposed aponeurosis, by palpation, it can be decided whether or not a radical procedure is possible and the extent of such a procedure

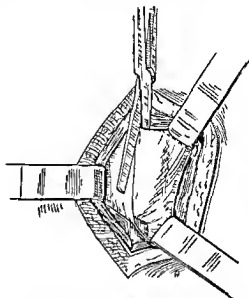


Fig 171—Posterior portion of thyroid cartilage is being removed by means of heavy scissors

The Operation—*Anesthesia*—Avertin (65 mg) is administered as a basic anesthetic reinforced by ether. If surgical diathermy is to be used chloroform must take the place of ether

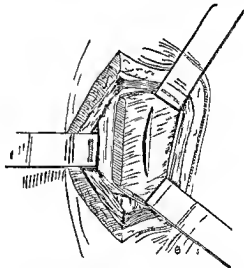


Fig 172—Shows internal perichondrium and laryngopharyngeal aponeurosis with incision into pharyngeal cavity

and all precautions must be used. It is always necessary to do a preliminary tracheotomy, this differs somewhat from the general type of tracheotomy. First, a transverse incision should be made as small as possible, thereby preserving

as much of the skin as possible for future turning in of a flap if necessary for the operation of pharyngotomy. Second the thyroid isthmus is never retracted but if met with during the course of the operation it is cut through. Thirdly a disk of trachea is removed thus allows quick healing of the tracheotomic wound at time of decannulation.

Glands—Usually some nodes in the neck are palpable on the affected side when so a complete block dissection is done including removal of jugular vein ligation and severing of the external carotid artery the submaxillary gland and lymph nodes are removed. The sternoclei-

by means of heavy scissors the posterior portion of the hyoid bone and thyroid alae are removed. This brings one down on to the laryngopharyngeal aponeurosis then by palpation the extent of the growth may be ascertained. The pharynx is opened care being taken to protect the surrounding area from contamination by pharyngeal mucus. The growth is then removed with an adequate margin. Bleeding if any is controlled. Laryngopharyngeal aponeurosis is brought together carefully by sutures the infrahyoid muscles are then sutured over the line of incision. The rest of the wound is left open and a nasal feeding tube is inserted.



Fig 173—The skin and platysma are retracted. The cervical fascia, superficial lymphatic glands and submaxillary gland have been removed. The anterior edge of the sternomastoid has been retracted outward to expose the structures lying beneath it. The carotid sheath is opened showing the vessels.

domastoid muscle however is reserved to cover over the deep vessels of the neck by suturing the muscle to the prevertebral fascia. This area is drained posteriorly by a separate puncture.

Pharyngotomy (Figs 168-172)—A vertical incision is made from the lateral cornua of the hyoid bone extending downward over the lateral aspect of the thyroid alae including the infrahyoid muscles down to the thyroid cartilage. The infrahyoid muscles are retracted forward and backward so as to completely expose the posterior portions of the great cornua of the hyoid bone and thyroid alae. The structures on the under surface of these are separated then

This description is for the very early case of epiglaryngeal carcinoma.

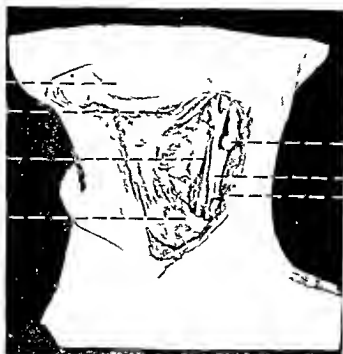
In most of these patients however by the time they are examined the growth is so extensive that the simple type of pharyngotomy is inadequate and a more extensive operation becomes necessary. An additional incision is made more or less at right angles to the original one running from the angle of the lower jaw downward forward and upward to the symphysis mandibulae. These flaps and the platysma are turned back the superficial fascia the submaxillary gland and the deep fascia are likewise removed upward and backward as far as

SKIN AND PLATYSMA
RETRACTED

SUBMAXILLARY FOSSA
WITH GLAND REMOVED

POSTERIOR EDGE OF
THYROID CARTILAGE

OMOHYOID MUSCLE



INTERNAL JUGULAR
RESECTED AND
LIGATED

CAROTID VESSELS

STERNOMASTOID
MUSCLE

Fig 174 —The deep cervical fascia is removed and the internal jugular vein ligated and resected. All veins and arteries coming off anteriorly have been ligated and cut. The pneumogastric nerve is seen. The thyrohyoid muscle is cut exposing the thyrohyoid membrane. The inferior constrictor has been separated from the thyroid cartilage. The hyoid bone is also exposed.

CORNUA OF THE
HYOID BONE

THYROHYOID
MEMBRANE

THYROID CARTILAGE

MUSCLES COVERING
THYROID CARTILAGE
RETRACTED



STERNOMASTOID
MUSCLE SUTURED TO
THE PREVERTEBRAL
FASCIA

CONSTRICTOR MUSCLE
SEPARATED FROM THE
POSTERIOR EDGE OF
THYROID CARTILAGE

Fig 175 —The anterior edge of the sternomastoid is sutured to the prevertebral fascia covering and protecting the great vessels. The area is drained by a counter opening posteriorly. The muscles have been reflected from the hyoid bone and the thyroid cartilage. The constrictors of the pharynx are reflected, and the great cornu of the hyoid bone and thyroid cartilage exposed for removal.

the spinal accessory nerve and posterior triangle. This step exposes the deep vessels of the

The internal jugular is ligated and resected. The external carotid may or may not be ligated. The



Fig 176—The great cornu of the hyoid bone is removed as well as the greater half of the thyroid cartilage; the wall of the pharynx is thus well exposed and ready for incision.

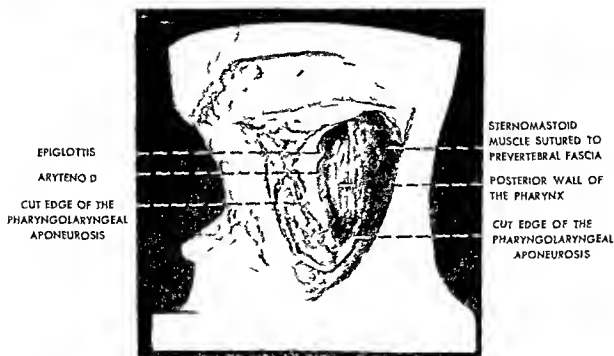


Fig 177—The pharynx is opened, exposing the base of the tongue, epiglottis, arytenoids, and postcricoid regions. This gives an excellent approach to a growth in either the epiglarynx or the hypopharynx.

neck and the muscles surrounding the larynx (Fig 173). All the vessels that come off anteriorly from the great vessels are ligated and cut.

The sternomastoid may or may not be removed depending on the glandular involvement (Fig 174).

The next step is to suture the sternomastoid to the prevertebral fascia, this covers the great vessels and thus protects them from infection when the pharynx is opened. The area is drained by a counter opening posteriorly (Fig 175). The following muscles are then reflected from the

defect. The picture (Fig 178) shows a flap of skin turned in to make up a defect in a post-cricoid carcinoma. A feeding tube is inserted into the esophagus. Later, a plastic operation is performed to close the neck. If the growth is on the anterior wall of the esophagus the flap will



Fig 178—Closure for post-cricoid growth when it has become necessary to turn in a flap of skin from the neck to make good the defect in the pharynx. This gutter is closed later by a plastic flap.

hyoid bone and thyroid cartilage, the hyoglossal, thyrohyoid, sternohyoid, sternothyroid, stylopharyngeus, and the inferior and middle constrictors of the pharynx. One is then ready to remove, by means of heavy scissors, the exposed portions of the hyoid bone and the greater portion of the thyroid cartilage (Fig 175). This permits one to palpate the growth through the pharyngeal aponeurosis and mucous membrane, and to decide where to make the opening into the pharynx so as to be well around the malignant area, or if the growth is too large to remove, one may hack out, so to speak, and not proceed any further (Fig 176). The next step is to open the pharynx by an incision which gives an approach to either a hypopharyngeal or an epiglottic growth, and which may be continued upward or downward as the case may be (Fig 177).

Before the operation, one has a fair idea of how much tissue it will be necessary to remove, and the flap of skin at the time of the original incision is made sufficiently large to fill the

defect and be turned in. If the growth occurs posteriorly the flap is turned in over the muscles and vessels.

HENRY BOYLAN ORTON

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BENIGN TUMORS OF THE NASOPHARYNX

Types—Benign tumors of the nasopharynx are any nonmalignant tumefaction or enlargement of tissues encroaching upon the normal lumen and by so doing eventually interfering with its normal relations and functions. Many different types of tissue have been reported in these growths. Excluding adenoidal hypertrophy a fairly complete list of these tumors would include fibroma, hemangioma, neurofibroma, lipoma, xanthoma, dermoid, teratoma, chondroma, mixed tumor, and several types of cystic and polypoid structures. Of these the most common is fibroma.

Nasopharyngeal Fibroid Tumor—These tumors usually occurring in young adults are comparatively rare but occur more often than any of the other types.

Etiology—There is no known cause for the development of these lesions which arise from the periosteum of the bony structures making up the walls of the nasopharynx. They may be single or multiple, sessile or pedunculated. They are said to develop a few years prior to and during the years of puberty and to have a tendency to degenerate in the mid twenties. Many observers including the author doubt the latter part of this statement. At any rate it certainly does not always apply. These lesions often are the cause of disorders of sufficient gravity to render their obliteration or removal imperative.

Pathology—Numerous blood vessels and much dense fibrous tissue with few cells make up the microscopic picture of the adult tumor. In earlier growths cellular deposits may be numerous somewhat resembling sarcoma.

Symptoms—Bleeding and obstruction are usually the earliest symptoms. In some cases the tendency to bleed is slight and occurs only when there is manipulation of the mass. In others severe profuse spontaneous hemorrhage may frequently occur. Although this tumor does not invade tissue it can erode bone by pressure and therefore upon enlargement can produce disorders in organs adjacent to the nasopharynx. Ear symptoms are common. These range from a sensation of fullness, tinnitus and dull pain to varying degrees of deafness and otitis media. Purulent nasal discharge results from improper sinus ventilation and drainage. The adenoid voice and facies are com-

mon. If lateral pressure is exerted upon the base of the skull there will be pain over the trigeminal distribution. Dysphagia may result from pushing downward of the palate. Hemorrhage, sepsis, profound secondary anemia and meningitis have been observed as causes of death in extreme cases of this type of growth.

Diagnosis—Anterior rhinoscopy, nasopharyngeal mirror studies, digital examination of the nasopharynx and careful inspection of the cavity with the nasopharyngoscope are all necessary for the proper study of all types of nasopharyngeal lesions. Even then no one can be sure of the histological nature of the growth. Punch biopsy specimens should always be secured. This rule should apply to all lesions of the nasopharynx where abnormal tissue even in small amounts seems to be present. On several occasions the author has found tissue in the fossa of Rosenmüller which explained the presence of malignant cervical glands. These biopsies should be a hospital procedure as packing is often necessary to control bleeding. If the growth is not completely obstructive the nasopharyngoscope may be inserted in one nostril and the punch in the other. By this means one can secure material from any desired location.

Treatment—Nonsurgical methods of treatment such as the application of radium in needle or capsule form supplemented by roentgen ray therapy may be tried. In the hands of the author this has never been successful. Surgical removal has always finally been necessary. The difficulties of this procedure are those of approach and control of hemorrhage. Intratracheal or rectal anesthesia should be used and the nasopharynx exposed by splitting the palate. It is usually necessary to remove some of the bone of the hard palate by rongeurs to secure adequate exposure. The larger masses are then removed by snare and the bases thoroughly treated by electrodesiccation. Following this method the author has had neither recurrence nor serious hemorrhage. Adequate packing and carotid ligation should be used when bleeding shows a tendency to be uncontrollable. Supportive transfusions should be used when indicated. Periodic follow up examinations should be carried out after surgical removal and electrocoagulation used early on any areas where recurrence seems to be taking place.

Hemangioma—These lesions show the gross and microscopic picture of this type of growth found elsewhere. They may be primary in the

nasopharynx or extensions from lesions in the nose or posterior pharyngeal wall. Although not malignant by metastasis they have a tendency to enlarge and invade adjacent structures. This and their high vascularity make them potentially dangerous. Radium and roentgen ray therapy are the treatments of choice. Surgery should be used when these are unsuccessful.

Neurofibroma and Mixed Tumors.—These most often occur embedded in the tissues of the posterior pharyngeal wall. Enlargement causes obstructive symptoms in the pharynx and nasopharynx. Biopsy is difficult due to the depth of the tissue characteristic of the lesion. All of the author's cases were first considered to be sarcomas. These growths are well encapsulated and yield only to surgical removal. Under intra-tracheal anesthesia they can be removed with relative ease by incision of the posterior pharyngeal wall. The masses are quite large and often extend as high as the base of the skull. In one of the author's cases the internal carotid artery lay upon the inner surface of the mass and required ligation.

Other Benign Tumors.—Isolated cases of the other tissues mentioned have been reported. Their true identity as a rule is not known until removal has been effected and the tissue subjected to microscopic study. Although true polyps may arise in the nasopharynx, the most usual source of these growths is the pedunculated nasal polyp with stalk long enough to allow it to reach the nasopharyngeal cavity. Occasionally they can be seen protruding below the lower border of the soft palate. If they are quite old they may be so fibrotic as to be mistaken for a true fibroma. Surgical removal is necessary in these cases. Cysts of residual adenoid tissue or the pharyngeal bursa may be clinically significant as areas of focal infection. They, too, demand surgical care.

In dealing with the nasopharynx one should always subject this space to careful inspection especially with the nasopharyngoscope. Punch biopsy specimens should be taken of any tissue abnormal in appearance.

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CANCER OF THE NASOPHARYNX

Cancer of the nasopharynx, although not the most frequent, is one of the most malignant growths of the upper respiratory and alimentary tracts. It was not clearly recognized as an anatomic entity before the beginning of the present century. Even today the diagnosis is not made in a high percentage of cases because the symptoms of metastases frequently dominate the clinical picture. In a cancer clinic, about one half of the patients with this disease are referred because of signs of metastases alone without a correct diagnosis of the location of the primary tumor.

The term *cancer of the nasopharynx* includes all malignant growths arising on the walls of this cavity and should not include nasal cavity growths. Cancer in the nasopharynx occurs most often on the posterior wall, in the region of the nasopharyngeal tonsil with its lateral extensions into the recessus pharyngis (fossa of Rosenmüller), and next in frequency on the lateral walls, on the ridge (torus tubarius) which surrounds the orifice of the eustachian tube. Occasionally, a growth may originate near the junction of the nasal and oral pharynxes. The floor of the nasopharynx (the upper surface of the soft palate) is practically never the site of origin of cancer.

The *lymphatics* of the nasopharynx are numerous. They originate mainly in the pharyngeal tonsil (adenoid) and run laterally and downward on the pharyngeal aponeurosis, some of them terminating in the median and lateral retropharyngeal lymph nodes (Fig. 179). The collecting trunks terminate for the most part in the upper nodes of the spinal accessory chain, which lie under the upper end of the sternomastoid muscle, and, also, in the subdiaphragmatic node of the internal jugular chain. From these nodes efferent branches run down to the middle and lower groups of the internal jugular and spinal accessory chains. Although not emphasized by anatomists, it is obvious that the cervical lymphatic system must contain many by-passes which provide routes through

which metastases may leave the primary nasopharyngeal tumor and appear first in a lymph node in the middle or lower portion of the neck

Incidence—Cancer of the nasopharynx is undoubtedly much more common than reports in the literature indicate. Nasopharyngeal cancer is so often included with growths of the nasal cavity and of the pharynx as a whole by many authors that the figures even in the best reports are unreliable in regard to the nasopharynx itself. In the Head and Neck Clinic at Memorial Hospital cancer of the nasopharynx makes up about 2 per cent of all malignant growths.

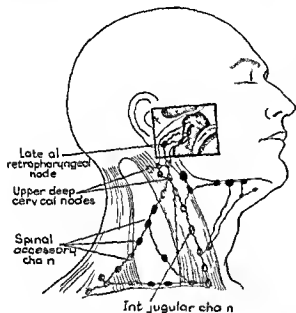


Fig. 179—Diagram of lymphatics of the nasopharynx. The collecting lymph trunks in the nasopharynx pass in part through the retropharyngeal node but mainly directly to the upper deep cervical lymphatics and then drain downward along either the internal jugular vein or the spinal accessory chain.

The unusual susceptibility of the Chinese and other Asiatics to this disease has been emphasized by many investigators. The fact that the disease occurs with such frequency in Orientals who live in a fairly normal American environment suggests that the tendency is racially inherited and not due to either hygienic or dietary factors.

Age and Sex—Cancer of the nasopharynx occurs at an earlier age and is found more often in children and at ages below thirty than any other malignant growth of the upper respiratory and alimentary tracts. About one fifth of the patients with cancer of the nasopharynx are under the age of thirty. As with most other

forms of cancer in the head and neck nasopharyngeal cancer is predominantly a disease of the male (about 80 per cent).

Causative Factors—The more common forms of chronic irritation which are known to produce definite precancerous changes in the oral mucous membranes apparently play no role in the causation of nasopharyngeal cancer. Leukoplakia, syphilis, tobacco, frequent upper respiratory infections, allergy or abnormalities of the lymphoid tissues of Waldeyer's ring are of no etiologic significance nor is it possible to demonstrate any specific causative factors for cancer in this locality.

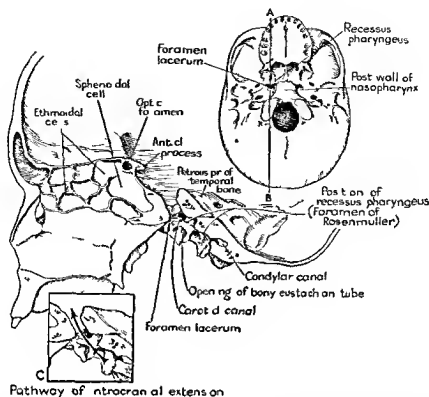
Pathology—A histologic classification of malignant tumors of the nasopharynx is given in the following table.

HISTOLOGIC TYPES OF CARCINOMA OF THE NASOPHARYNX IN A SERIES OF 87 CASES OBSERVED AT MEMORIAL HOSPITAL

	No.	Total
A Epidermoid carcinoma		73
(a) Squamous cell carcinoma	5	
(b) Transitional cell carcinoma including the anaplastic varieties	42	
(c) Lympho-epithelioma	14	
(d) Spindle cell carcinoma	4	
(e) Not definitely classified	8	
B Lymphosarcoma		11
C Malignant tumor of salivary gland type		3
Total		87

Since cancer in this region arises most commonly at the site of the pharyngeal tonsil, it is natural that highly anaplastic epidermoid carcinomas and lymphosarcomas should make up a large proportion of the total. As is also found with the palatine tonsil, minor salivary glands are present in the mucous membrane lining the walls of the nasopharynx so that the occurrence of adenocarcinoma is also to be expected.

Symptoms, Morbid Anatomy, and Clinical Course—The primary lesion in the nasopharynx causes few if any symptoms in the early stages. In the order of their frequency the first symptoms are cervical metastases, nasal obstruction, headaches or local pain, and defective hearing or pain in the ear. The nasopharynx, a cavity with rigid walls, is much wider than the nasal cavities in front or the glottis below. Obstruction to breathing will not occur therefore except in the later stages when the tumor has become large and bulky. In the majority of such cases cervical metastases, unsuspected by the patient, are also found. Nasal



Pathway of intracranial extension

Fig 180—The fossa of Rosenmüller lies immediately over the foramen lacerum which gives direct access for the intracranial extension of nasopharyngeal cancer. The large diagram is a sagittal section along the line A B through the foramen lacerum, illustrating the juxtaposition of the lateral wall of the nasopharynx, the foramen lacerum, and the cranial cavity. Insert C demonstrates the direct pathway of this extension.

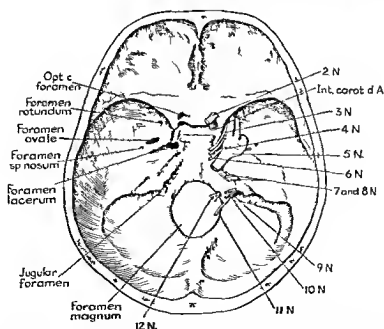


Fig 181—Diagrammatic representation of the base of the skull internally showing the juxtaposition of the foramen lacerum and the third, fourth, fifth, and sixth cranial nerves. Nasopharyngeal cancer extending intracranially through the foramen lacerum comes in contact first with the sixth cranial nerve. The growth may then spread forward through the superior orbital fissure into the orbit or backward into the posterior fossa to involve all the cranial nerves.

discharge and bleeding are usually late symptoms. When the growth arises in or near the orifice of the eustachian tube, deafness occurs before nasal obstruction. Discomfort in the ear and disturbances in hearing are common as late symptoms, especially following irradiation.

Intracranial and Intra orbital Extension—A characteristic feature of nasopharyngeal cancer is its tendency toward extension intracranially and to the orbit. Even if the growth does not arise directly in the fossa of Rosenmüller, one of these fossae is practically always involved early in the course of this disease. Since this fossa lies directly under the foramen lacerum (Fig 180), cancer may extend by direct continuity of soft tissues from the nasopharynx through the foramen lacerum and into the cranium, a distance of about 1 cm, without erosion of the bone. In the later stages of intracranial extension, when several cranial nerves are paralyzed, it is almost always possible to demonstrate roentgenographically an erosion of the bony floor of the middle fossa of the skull. The presence of an infected growth, often heavily irradiated, almost invariably produces some demonstrable changes in the adjacent bone, which in many cases do not represent erosion by cancer. Direct invasion by bone erosion is not initially the cause of intracranial extension of the disease. The foramen lacerum permits direct intracranial extension into the extradural space without the necessity of bone erosion. The growths remain extradural and subperiosteal because the dura forms the internal periosteum of the skull. Once the disease has extended through the foramen lacerum, the surrounding bone may be attacked from without and within, being the result rather than the cause of intracranial extension.

In addition to localized pain, diplopia, and vomiting, the symptoms of such extension are progressive unilateral paralysis of the cranial nerves. The sixth nerve passes directly over the foramen lacerum (Fig 181) and is, therefore, the first cranial nerve to be involved by extension of the cancer. The third, fourth, and fifth cranial nerves are next involved in frequency. When the disease extends into the orbit there is pressure on the second nerve. The branches from the seventh to the twelfth cranial nerves, which lie further posteriorly, may be involved in the later stages of intracranial extension.

Invasion of the orbit occurs in about 4 per cent of the cases. The tumor in advancing

through the foramen lacerum comes in contact with the internal carotid artery, lying in the carotid groove. It grows along this groove and enters the orbit through the superior orbital fissure. The second, third, and fourth cranial nerves enter the orbit through this fissure and may become involved at this point, if not already involved at the foramen lacerum. With invasion of the orbit, there are exophthalmos, ophthalmoplegia, eventual blindness, and gradually increasing local pain, later diffused to the whole cranium of the affected side. Rarely, the growth may arise within or possibly extend through the eustachian tube, so as to be present both in the nasopharynx and in the ear by perforation of the tympanic membrane.

Metastases—Metastasis to the lymph nodes of the neck occurs in about 80 per cent of the cases and is the first and only complaint in one half of them. The cervical lymph nodes which are most frequently involved are those lying beneath the mastoid tip under the upper end of the sternomastoid muscle (upper nodes of the spinal accessory chain). Next in frequency are those situated over the carotid bulb. After invasion of the subclavicular node, metastasis extends to the lower portions of the neck with more frequent dissemination to the posterior triangle than occurs in cancer of the oral cavity. The submaxillary group of lymph nodes is rarely the initial site of metastasis. Widespread bilateral cervical node enlargement is characteristic of the later stages of the disease. Metastatic involvement of the lateral retropharyngeal lymph nodes may occur.

Systemic metastases are more frequent with cancer of the nasopharynx than with most other growths of the upper respiratory and alimentary tracts. Dissemination below the clavicle occurs eventually in one third of the patients. The most usual form of generalized metastasis is skeletal. The liver and lungs are next involved in frequency, these viscera tend to be invaded in the later stages of all uncontrolled cases.

Diagnosis—There is probably no other form of cancer in which the anatomic diagnosis is more often delayed or entirely missed than in cancer of the nasopharynx. This fault may in part be attributed to the fact that only in about one half of the cases are there any early local symptoms from the primary lesion. In addition, except for cancer, the nasopharynx is a relatively unimportant structure from the standpoint of disease and it is, therefore, usually

neglected in the course of an average physical examination. Progressive unilateral deafness or unexplained cervical node enlargement in the adult should suggest the possibility of nasopharyngeal cancer. In the adult, enlarged cervical nodes are almost always malignant and usually metastatic from a primary tumor in the upper alimentary or respiratory tracts, in these cases the nasopharynx is the most likely site for a primary cancer.

The most efficient method of examining the nasopharynx is by using a throat mirror in the pharynx while depressing the tongue and retracting forward the free edge of the palate. Endoscopic examination (nasopharyngoscope,

grown by hypertrophic lymphoid or granulation tissue as in the nasopharynx. A straight biopsy forceps inserted directly backward through the nasal cavity or a curved forceps through the open mouth may be used to remove a specimen from a nasopharyngeal tumor (Fig 182). The manipulations should in either instance be guided visually by a mirror or palpably by a finger.

Adolescent nasopharyngeal fibroma or the rare case of tuberculosis of a retropharyngeal lymph node might be clinically confused with cancer, but microscopic examination of a biopsy specimen will establish the correct diagnosis.

Biopsy

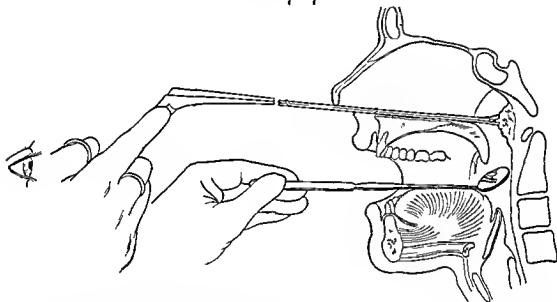


Fig 182—All parts of the nasopharynx are best examined with the aid of a mirror inserted through the mouth into the oropharynx. A biopsy specimen from this region may be removed through the nasal cavity, the operator being guided either visually by the mirror or palpably by a finger.

antroscope, etc.) reveals only a limited portion of the nasopharyngeal walls because of the rigid system of lenses. The primary lesion in this area may consist only of a slight irregularity or nodularity which may or may not be ulcerated. The fossa of Rosenmüller may present irregularities or may be partially obliterated. A biopsy of tissue from any suspicious areas is indicated in any case in which cancer of the nasopharynx might be considered as a possible diagnosis. A single negative biopsy report should not be considered conclusive in the presence of a clinically positive tumor, for in no other portion of the upper respiratory and alimentary tracts is cancer so apt to be over-

Treatment.—The nasopharynx is comparatively inaccessible to surgical exposure, the area is extremely vascular, the growths are poorly delimited, and there is frequent early extension of the disease intracranially and to the orbit. For practical purposes, therefore, the primary lesion in the nasopharynx must be considered unsuitable for adequate surgical removal. On the other hand, nasopharyngeal cancer is one of the most radiosensitive of all tumors of the upper respiratory and alimentary tracts and this region tolerates heavy doses of radiation. For these reasons, irradiation is preferable to surgical removal for treatment of the primary tumor.

Irradiation—Irradiation of the nasopharynx is best carried out by a combination of external irradiation through the cheeks (malar portals) and intracavitary radiation by radium or radon. Occasionally, supplementary treatment to the primary tumor may be given perorally through

tionated roentgen irradiation through the cheeks, using the following factors: 250 KV, target skin distance of 50 to 60 cm, 1.5 mm of copper filtration and circular ports of 5 to 6 cm in diameter. The daily dose should be 300 to 400 r applied to each malar portal in rotation

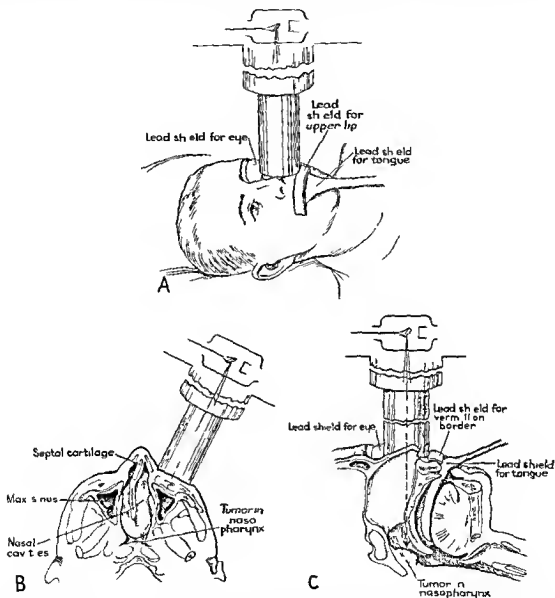


Fig 183—A, External irradiation of cancer of the nasopharynx through the cheek. One lead shield protects the eye, a second, the tongue and a third, the vermilion border of the upper lip. B, External irradiation to the nasopharynx shown diagrammatically by a horizontal section. The beam is directed through the cheek inward and backward at an angle of about 35 degrees from the sagittal plane. C, External irradiation through the cheeks to the nasopharynx, showing the position of the lead shields on the tongue, the upper lip, and the eye.

the palate. Although cancer lethal doses of radiation can undoubtedly be delivered to the nasopharynx by either one of these methods alone, a large proportion of the undesirable effects of either method can be obviated by a combination of the two.

The treatment is ordinarily begun by frac-

for a total dose of 3500 to 4000 r times 2, interrupted by the application of intracavitary radiation. The centering of the portals and the shielding should be so designed as to avoid unnecessary irradiation to the eye, tongue, and the vermilion border of the lip (Fig 183, A, B, C). When irradiating the nasopharynx through

malar portals, it is important to place a lead shield on top of the tongue, otherwise the upper

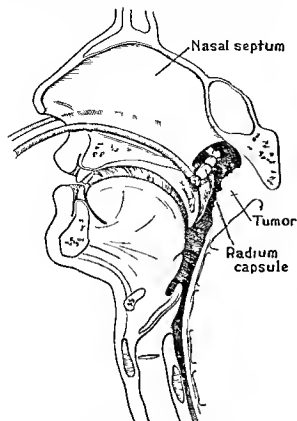


Fig 184.—Intracavitary irradiation of cancer of the nasopharynx by a capsule of radium or radon tied in the center of a rubber catheter which is threaded into one nasal cavity and out the other. Although this method has the advantage of being simple and requiring no special apparatus it is disadvantageous in that the capsule tends to lie on the upper surface of the soft palate rather than in contact with the tumor itself.

edge of the beam will produce a painful radiation reaction on the dorsum of the tongue.

To spare the skin of the cheeks and the nasal cavity from too marked effects of irradiation part of the external radiation may be given perorally through the palate provided the patient is able to open the mouth widely so that a peroral cylinder of sufficient size can be inserted. A cylinder of at least 4 cm in diameter with an oblique end should be used and the direction of the beam carefully checked so that the lesion is accurately centered. The total dose is somewhat variable but with the portal size mentioned and a distance of 50 cm a daily dose of about 400 r may be given in place of the usual dose to one of the cheek portals.

Intracavitary Radiation—By combining gamma with roentgen radiation the untoward effects of either of these agents used alone may be partially overcome. After the application of about 1500 to 2000 r of roentgen irradiation to each cheek the treatment is interrupted and intracavitary radon is applied for a dose of about 400 to 500 millicurie hours, the skin in the meantime being given a four to five day rest. On completion of this intracavitary dose roentgen irradiation is resumed until a total dose of about 3500 to 4000 r has been completed to each cheek. In refractory cases, the intracavitary radon capsule can then be reinserted for an additional dose of 250 to 400 millicurie hours, but the total dose of intracavitary radiation should not exceed 1000 millicurie hours. Radium or radon may be intro-

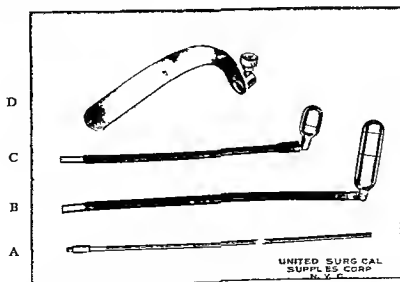


Fig 185.—Blady nasopharyngeal radium applicator. A, Steel shaft for inserting the applicator. B and C, Radon capsules with attached shafts. D, Immobilizing apparatus.

duced into the nasopharynx by tying a capsule into a rubber tube (Fig 184) or by an improved applicator which has been designed in this clinic (Fig 185). The technic of using the special nasopharyngeal applicator is illustrated in Figures 186 187 and 188

Treatment of Cervical Metastases—Since cervical metastases occur in about three fourths of all cases their successful treatment is one of the most important determining factors in the

controlling a fair percentage of nasopharyngeal cervical metastases

The irradiated areas must be limited otherwise the general systemic effect may prove fatal. Irradiation of the entire neck on the theory that

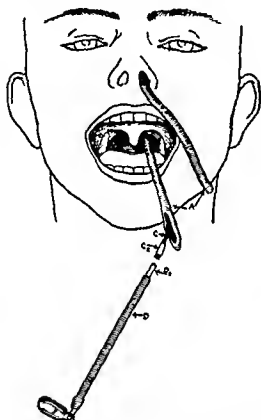


Fig 186—Insertion of the Blady nasopharyngeal applicator. A catheter (A) sheathing the flexible metal shaft (C) is threaded through the nasal cavity into the pharynx and out of the mouth. The nasopharyngeal applicator (D) is then attached and drawn into the nasopharynx so that the shaft protrudes from the anterior nostril.

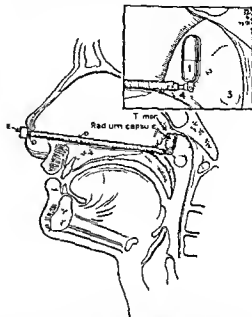


Fig 187—Blady nasopharyngeal applicator in place with a diagrammatic representation of the radon capsule in various positions.

even though nodes are not palpable the dissemination is probably widespread would be sound if cancer lethal doses could be given safely to the entire potential lymph node bearing areas of the neck. Radiation of such extent

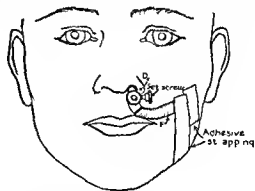


Fig 188—The applicator (D) held in place by the immobilizing attachment (F) which is fixed to the cheek by adhesive tape.

prognosis of this disease Neck dissection is of little value where a highly malignant and plastic radiosensitive growth tends to metastasize early and widely to both sides of the neck. The problem here is more difficult than with slowly growing and more highly differentiated tumors of the lips, tongue, or cheeks in which the nodes more often tend to appear late in the course of the disease and to be unilateral and limited in extent. In contrast to surgical intervention, irradiation is capable of permanently

sive areas produces untoward sequelae the patient if he survives is so uncomfortable and disabled as to render this treatment of little ultimate value. The size of the portal should be limited to the location and extent of palpable

or otherwise demonstrable involvement by cancer. In this way the tissue dose may be increased so as to produce a lethal effect on the tumor. The most successful treatment consists of a combination of fractionated roentgen irradiation and implantation of radon seeds. Supplementary radium therapy obviates the necessity of pushing the dose of roentgen radiation beyond its judicious limit.

The actual technic depends on the morbid anatomy of the metastasis. In conjunction with irradiation of the primary nasopharyngeal tumor, the neck portals, usually the upper deep cervical (7 to 8 cm. in diameter), are irradiated daily, alternately, for a dose of about 3000 r to each portal. It is then preferable to stop short of the maximum dose and to implant gold radon seeds through the skin for a tissue dose

Radiation sickness, therefore, is a common complication. There may also be some damage to the base of the brain and partial derangement in the function of the various glandular, vascular, and neural structures of the neck. Undesirable radiation effects may be considerably reduced by not using unnecessarily large portals. Adequate vitamin therapy, especially with the B complex, is of distinct value in the treatment of radiation sickness. When the epidermis has broken down, the area should be kept covered by a bland ointment spread on fine mesh gauze. Mucosal reactions progress to a membranous mucositis, involving the nasopharynx, nasal cavities, the under surface of the soft palate, and occasionally the pharynx. These areas should be frequently irrigated with a nasal syphon. Following irrigations, crusts and clots

MILLICURIES IN GOLD SEEDS REQUIRED TO DELIVER SPECIFIED DOSES TO MASSES OF VARIOUS DIAMETERS

Skin Erythema Doses	Millicuries for Masses with Diameters of											
	1 0 Cm	1 5 Cm	2 0 Cm	2 5 Cm	3 0 Cm	3 5 Cm	4 0 Cm	4 5 Cm	5 0 Cm	6 0 Cm	7 0 Cm	8 0 Cm
5	1 0	2 5	4 0	7 5	10	12	14	17	20	27	35	45
6	1 2	3 0	4 8	9 0	12	14	17	20	24	32	42	54
7	1 4	3 5	5 6	10 0	14	17	20	24	28	38	49	63
8	1 6	4 0	6 4	12 0	16	19	23	27	32	43	56	72
9	1 8	4 5	7 2	14 0	18	22	26	31	36	49	63	81
10	2 0	5 0	8 0	15 0	20	24	29	34	40	54	70	90
11	2 2	5 5	8 8	17 0	22	26	32	37	44	59	77	99
12	2 4	6 0	9 6	18 0	24	29	35	41	48	65	84	108

of 6 to 7 erythema doses (see table above). Roentgen irradiation is interrupted for seed implantation.

Treatment of Widely Disseminated Metastases—Since nasopharyngeal cancer is invariably highly radiosensitive, palliative treatment of widely disseminated metastases of this disease is distinctly worth while. Persistent pain, produced by skeletal lesions, can frequently be relieved by moderate doses of external radiation. Such metastases should be accurately localized so that an adequate dose can be delivered without too great a drain on the general tolerance. It is not ordinarily profitable to irradiate pulmonary metastatic lesions.

Complications of Treatment—In the treatment of nasopharyngeal cancer a relatively large volume of tissue must be irradiated

may be removed with forceps and the cavity sprayed with liquid petrolatum. No radiation reaction should occur in the eyelids, conjunctiva, tongue, or lips if proper shielding has been carried out.

Prognosis—Nasopharyngeal cancer is by no means a hopeless disease if properly treated by aggressive irradiation and the end result is about the same as in cancer of the tongue. The prognosis is least favorable in the young and in the very old. In young subjects, especially in children, systemic metastases appear almost invariably, even though the response of the primary tumor to treatment is favorable. The prognosis in the female is slightly better than in the male. The presence of metastases is one of the most important factors in the prognosis, as shown in the following table.

FACTORS INFLUENCING THE FIVE YEAR CURE RATE IN 87 CASES OF CANCER OF THE NASOPHARYNX OBSERVED AT MEMORIAL HOSPITAL FROM 1930 TO 1934

	Total Number of Cases	Number of 5-Year Cures	Percentage of 5 Year Cures
Age in years			
Below 40	32	7	22
40 to 49	21	5	24
50 to 59	19	6	32
Over 60	15	2	13
Sex			
Males	68	15	22
Females	19	5	26
Metastases			
None at any time	15	7	47
None on admission	18	7	39
Present on admission	62	13	21
Sometime during course	65	13	20
Developed after admission	3	0	0
Bilateral metastases	17	4	24
Position of cervical nodes involved			
Upper part of neck, one side	31	4	13
Upper part of neck, both sides	17	3	17
Entire neck one side	10	2	20
Carotid bulb, single node	7	4	57
Extension to the base of the skull			
On admission	3	0	0
Developed after admission	28	2	7
Histologic classification			
Lymphosarcoma	11	3	27
Malignant salivary gland tumor	3	0	0
Epidermoid carcinoma	73	17	23
Squamous carcinoma	5	1	20
Transitional cell carcinoma	42	10	24
Spindle cell carcinoma	4	0	0
Lympho-epithelioma	14	6	43
Not definitely classified	8		

In the Head and Neck Clinic at Memorial Hospital the net five year cure rate in patients without metastases at the time of admission is 50 per cent and the net five-year cure rate for all cases, including advanced stages, is 25 per cent

FIVE YEAR END RESULTS IN CASES OF CANCER OF THE NASOPHARYNX OBSERVED AT MEMORIAL HOSPITAL FROM 1930 TO 1934*

Total number of patients 87
Indeterminate group
 Dead as a result of other causes and without recurrence 4

Lost track of without recurrence 3
 Total 7
Determinate group (total number minus those of indeterminate group) 80
Failures
 Dead as a result of cancer 60
 Lost track of with disease (probably dead) 0
 Living with recurrence 0
 Total 60
Successful results
 Free from disease after five years or more 20
 Net five-year end results (20/80) 25 per cent

HAYES MARTIN

REFERENCES

*This series consists of the cases of all patients with histologically proved cancer of the nasopharynx, both early and advanced admitted during the specified period. Only those patients are excluded who, for any reason, were unable to return for treatment, palpation and observation in the outpatient department, and those who were lost track of within the first month after no more than one or two visits

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CHORDOMA OF THE NASOPHARYNX

A chordoma is a tumor developing from embryonic remains of the notochord. It is called also *ecchondrosis spheno occipitalis*, *ecchondrosis physalisfora*, *chordocarcinoma*, and *chordoma epithelioma*. It is an uncommon type of tumor.

Etiology—The cause of an autonomous new growth of tissue that is commonly called a tumor is unknown. The number of more or less well-supported theories is large. It is generally accepted that embryonal cells possess the factors essential for tumor growth to a greater degree than any other tissue cells. The base of the skull is one of the places where remnants of the chorda dorsalis, or notochord, commonly persist.

Pathology—Chordoma belongs to the connective tissue type of tumors. Because of their origin in chordal tissue they may have roots in bony tissue at the base of the skull and in the dura. Structurally, the primary tumors are composed of a rather homogenous basic staining matrix in which are groups of large vacuolated cells resembling somewhat the vacuolated cells of chondroma or myxochondroma. In one of our cases the specimen first removed closely simulated a chondrosarcoma. Histologically as well as clinically chordoma of the nasopharynx may be benign or malignant.

Examination—When small the tumor is seen in the mirror as a smooth or nodular mass covered with mucosa of normal appearance, except that in some cases branching vessels are visible. The sagittal fissures of adenoidal tissue are usually obliterated. At this stage palpation with the index finger shows the tumor to be rather firm and adherent to the basilar process of the occipital bone or to the wall of the sphenoidal sinus, or both. Some adenoid tissue may be felt on the surface or displaced to one side. A good direct view of the growth may be obtained by forcibly elevating the velum with a Yankauer nasopharyngeal speculum. At a later stage, the growth is usually ulcerated and then has the gross appearance of cancer of the nasopharynx.

Diagnosis—Examination shows the presence of a tumor. Its character can be determined only by biopsy. Before ulceration it has the gross appearance of fibroma, which is relatively common in this region, and of chondroma, which is not rare. Fibroma is obvious on microscopic examination. Atypical chondromas are difficult to differentiate histologically, but they can be ruled out when ulceration develops. As between a benign and a malignant chordoma no distinctions need be drawn because, as they occur in the nasopharynx, the initially benign growths are potentially malignant.

Treatment—If the roots extend into the skull and dura, as they usually do, surgical removal is impossible. The growths, being malignant or potentially so, should be treated in the same manner as cancer of the nasopharynx as described by Dr. Hayes Martin on preceding pages.

Prognosis is serious but not hopeless, except in the advanced cases.

CHEVALIER JACKSON

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PART III. THE EAR

ANATOMY OF THE EAR

The ear includes three anatomical divisions, namely, the outer, middle, and inner ear. The latter consists of the auditory cochlea, the vestibular utricle and saccule, and the semicircular canals. These connect with the central nervous system by the acoustic nerve which consists of two distinct bundles, namely, the cochlear and the vestibular nerves, entering the ear at the bottom of the internal auditory meatus. The outer ear, the middle ear, and the cochlea are functionally all parts of the acoustic system but will be described separately.

The External Ear.—The outer ear includes the auricle and the external auditory meatus. The *auricle* is made up, except in the lobule, of an irregular skeletal plate of elastic cartilage covered by skin. It is attached to the temporal bone by the anterior, superior, and posterior ligaments and by continuity with the cartilage of the acoustic meatus. The anterior, posterior, and superior auricular muscles attach to the auricle but grant little movement in man. The skin of the ear, continuous with that of the head, also helps hold the ear in position. On the anterior surface the skin adheres closely to the perichondrium of the cartilage, accounting for the frequent occurrence of hematomas from traumatic injury of this surface, but on the posterior surface it is looser.

The auricle has a relatively rich blood supply from the posterior auricular artery behind, and the superficial temporal artery in front. The veins are the auricular branches of the posterior auricular vein and the anterior auricular branches of the temporal vein. The superficial position of the vessels in the thin skin, which has little underlying subcutaneous tissue and fat, makes the auricle particularly susceptible to frost-bite. The lymphatics drain into the adjacent anterior and inferior auricular lymph nodes, and sometimes into the posterior auricular nodes.

The sensory innervation of the auricle is derived from branches of the great auricular and small occipital nerves, the auricular branch of

the vagus, and the auriculotemporal branch of the mandibular division of the trigeminus. The muscles receive their innervation from the posterior auricular and the temporal branches of the facial nerve.

The outer margin of the auricle is termed the *helix*. The *anthelix* lies parallel with it and forms a curved ridge which constitutes the posterior boundary of the deep depression, the *concha*, of the auricle. The *anthelix* divides superiorly into an anterior and a posterior crus, between which lies a shallow depression, the *triangular fossa*. A groove of varying depth, the *scapha*, separates the helix from the *anthelix*. Anterior to the meatus the *tragus* forms a backward projection which partly covers the opening. Just back of it lies the *antitragus*, separated from the *tragus* by a deep notch, the *incisura intertragica*, which also forms the inferior boundary of the *tragus*. The lobule is made up of connective tissue and fat, covered by skin. The *concha* leads into the external auditory meatus. The flaring opening of the meatus forms the *cavum conchae*, and the depression above this is termed the *cymba conchae*.

The *external auditory meatus* leads from the bottom of the *cavum conchae* to the tympanic membrane. The latter is so obliquely placed that the meatus is about 25 mm long on the superoposterior wall, and about 31 mm long on the antero inferior wall. The meatus has the form of an elongated and twisted S, the direction at first being upward, inward, and forward. It soon bends backward and inward, then downward, inward, and backward. To make it fully visible it is necessary to pull the auricle upward and backward. About 5 mm from the tympanic membrane the meatus shows a constriction called the *isthmus*, internal to which lies the *sulcus*. The outer cartilaginous meatus is supported by cartilage continuous with that of the auricle. This cartilage is divided by two horizontal clefts, the *incisurae santorini*, which may serve as pathways of drainage into the external meatus of abscesses of the parotid gland, or deep furuncles of the meatus may drain

through them into the parotid region. The inner portion, or osseous meatus, forms somewhat more than half of the canal. It begins at the downward and backward bend and is supported by a tubed process of the temporal bone. The osseous meatus is lacking at birth, but with growth of the head it is formed by enlargement of the neighboring bones and their fusion with the tympanic bone. The upper wall is formed by extension of the squamous bone, and the upper part of the posterior wall by expansion of the petrous bone during development of the mastoid process. This portion of the meatus lies adjacent to the mastoid cells, while the tympanic antrum lies along its wall just external to the attachment of the tympanic membrane. Cholestatomas of the antrum can thus break through into the external meatus, and infiltration of the periosteum just under the skin of the bony meatus may occur in mastoid infection. At the upper, posterior margin of the bony meatus the suprameatal spine forms a conspicuous landmark, to which is attached the superior ligament of the auricle.

The skin of the meatus lines the entire canal and forms the external layer of the tympanic membrane. In the cartilaginous meatus it is thick and contains numerous sebaceous and ceruminous glands, the latter forming an almost continuous layer. It is also provided with fine hairs. In the external part, especially in older men, the hairs may become large and stiff. In the medial, bony part the skin is thin and adheres closely to the periosteum. Hairs are lacking in this region and glands are present in only a small part of the posterior and superior walls. The anterior wall of the meatus lies in close relation to the temporomandibular joint, and part of it is separated from an upward extension of the parotid gland only by areolar tissue.

The Tympanic Membrane.—The tympanic membrane forms the inner boundary of the auditory meatus, separating the latter from the middle ear. It has a very oblique position, its outer surface being directed downward and forward and also laterally. The periphery of the membrane has a thickened border attached by a fibrocartilaginous ring to the tympanic sulcus of the temporal bone, save for about 5 mm in the anterosuperior part, where the sulcus is absent and forms the tympanic notch (notch of Rivinus). From the ends of the notch the anterior and posterior malleolar folds extend to the malleolar prominence, produced by the lateral

process of the malleus, and form a small triangular area covered by a thin, loose portion of the membrane, known as the flaccid portion, or Shrapnell's membrane, which is attached directly to the petrous bone. The greater part of the membrane, the tense portion, is concave, as seen from the meatus. The deepest point, known as the umbo, corresponds to the tip of the handle of the malleus. The antero-inferior quadrant is strongly illuminated when the membrane is examined by reflected light, and is known as the "cone of light." From the apex of the cone, at the umbo, the shadow formed by the handle of the malleus may be followed upward to the malleolar prominence.

The long axis of the membrane, measuring 9 to 10 mm, is nearly vertical. The short axis measures 8 to 9 mm. The ear drum usually makes an angle of about 140 degrees with the superior wall of the meatus. However, there is considerable variation in size and form, as well as in the angle. The membrane is about 0.1 mm thick and is composed of four layers: the cutaneous layer, the radiate fibrous layer (whose fibers are attached to the manubrium of the malleus and radiate outward to the fibrocartilaginous ring), the circular fibrous layer (whose fibers are most numerous near the circumference), and the mucous layer, a thin continuation of the mucous membrane of the middle ear. The two fibrous layers are absent in the pars flaccida, hence this is the weakest part of the membrane. Networks of elastic fibers are present, especially in the central part of the membrane and at its periphery. Along the manubrium of the malleus the skin is somewhat thickened and has a layer of subcutaneous tissue through which blood vessels and nerves reach the center of the membrane.

The external surface of the tympanic membrane receives its blood supply from tympanic branches of the deep auricular branch of the internal maxillary artery, while the internal mucous surface is supplied by branches from the anterior tympanic branch of the internal maxillary and the stylomastoid branch of the posterior auricular. The arteries of the two surfaces, however, anastomose with each other. The veins drain into those of the tympanic cavity and of the external meatus. Lymphatics occur in two sets, connected with each other. The nerves are derived from the auricular branch of the vagus, the tympanic branch of the glossopharyngeus, and the auriculotem-

poral branch of the mandibular nerve. The zone below the umbo is less sensitive and also less vascular than elsewhere, and does not lie near important structures of the middle ear. The pars flaccida is located near the ear ossicles and their ligaments and tendons of attachment. The chorda tympani also lies medial to the membrane above the level of the umbo.

The Middle Ear.—The middle ear includes the tympanic cavity, the tympanic antrum, and the auditory or eustachian tube. It is separated from the external ear by the tympanic membrane and from the inner ear by the membrane of the round window and the foot of the stapes

which contains the head of the malleus and the body of the incus. A recess in the posterior wall of the attic, known as the aditus, leads into the tympanic antrum. Bounded by Shrapnell's membrane laterally, the external ligament of the malleus above, and the neck of the malleus medially, is a space known as Prussak's space or pouch.

The roof is formed by a thin plate of bone, the tegmen tympani, which separates the attic from the middle cranial fossa. The petrosquamosal suture passes diagonally at its lateral margin. In the young, this suture transmits lymphatics and blood vessels from the middle ear

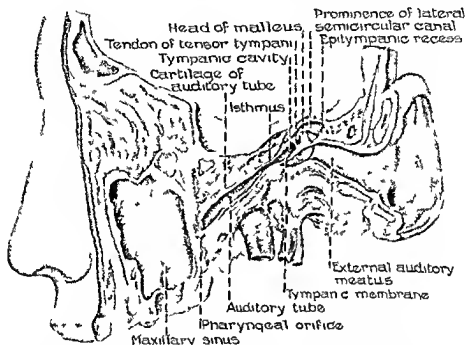


Fig. 189—Section showing relation of auditory (eustachian) tube to tympanic cavity and the external auditory meatus

in the oval window. Through the auditory tube it communicates with the pharynx, and through the tympanic antrum with the air cavities of the mastoid process.

The Tympanic Cavity.—The tympanic cavity is an irregular, obliquely placed, slitlike space within the temporal bone. Its vertical and anteroposterior axes are about 15 mm. long, while the transverse measurements are 2 mm. at the narrowest part, 4 mm. inferiorly, and 6 mm. superiorly. The main portion of the cavity lies between the tympanic membrane and the promontory formed by the labyrinth of the inner ear. It extends upward, however, as a vault-shaped space known as the attic or epitympanic recess,

to the middle cranial fossa. The plate is sometimes incompletely developed here to the extent that the mucous membrane of the tympanic cavity may come into direct contact with the dura mater. The floor of the main cavity continues downward as a deep groove, forming the hypotympanic recess. This begins below the level of attachment of the tympanic membrane. The superior bulb of the internal jugular vein lies just beneath the recess. The central part of the floor is thin and may show dehiscence. The anterior wall of the cavity continues directly into the bell-like opening of the eustachian tube. A thin plate of bone, bounding the tube medially, forms the lateral wall of the carotid canal.

Below the aditus in the posterior wall is a small conical projection the pyramid. This is hollow in part by a portion of the squamous bone which constitutes the upper wall of the external

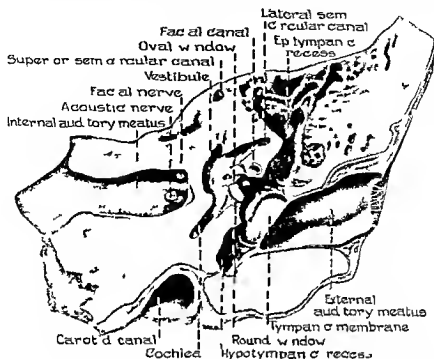


Fig 190 —Posterior view of section through the external auditory meatus, tympanic cavity, vestibule, and internal auditory meatus, right side.

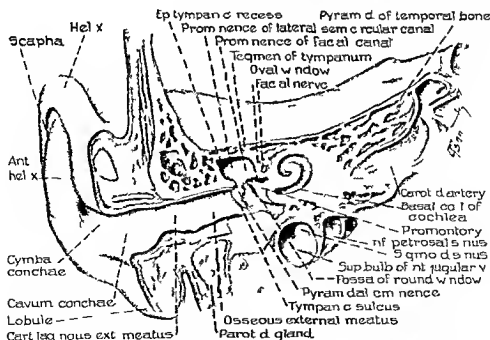


Fig 191 —Curved section through the external auditory meatus, tympanic cavity, cochlea, and pyramidal part of temporal bone, right side.

and houses the stapedius muscle whose tendon emerges through its tip. The lateral wall is formed chiefly by the tympanic membrane but meatus since the membrane does not reach the roof of the cavity. The medial wall is formed largely by the bony capsule of the labyrinth. At

about its middle the first turn of the cochlea constitutes the promontory. Two openings penetrate the wall into the labyrinth. The upper opening, the oval window, lies opposite the upper posterior quadrant of the tympanic membrane and above the posterior part of the promontory. It is occupied by the base of the stapes. Below the posterior part of the promontory the round window is covered by the secondary tympanic membrane which separates the cavity from the scala tympani of the cochlea. Above the promontory and the oval window a rounded ridge, formed by the bony canal of the facial nerve, arches backward and turns downward.

The Tympanic Antrum—The tympanic or mastoid antrum is a large air cell formed before birth by upward and posterior expansion of the tympanic cavity. It lies chiefly behind and above the osseous external meatus, but with wide variation both in size and position. In the newborn it lies almost directly above the bony meatus. As the mastoid process develops after birth the antrum moves gradually backward and downward. In the infant its lateral bony wall is 2 to 4 mm thick while in the adult it may have a thickness of 10 to 15 mm.

Later in fetal life cavities continuous with the antrum begin to project backward into the

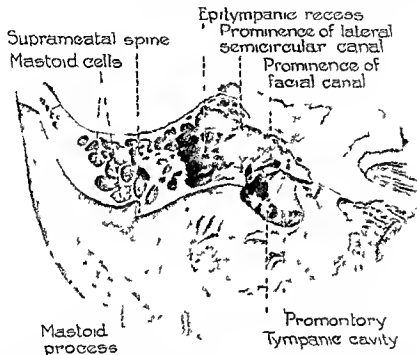


Fig. 192.—Section through upper posterior wall of the external auditory meatus showing relation of mastoid cells and epitympanic recess to the meatus and the relation of the epitympanic recess to the tympanum.

posteriorly in the medial wall of the aditus. The semicanal for the tensor tympani muscle lies just anterior to the facial canal and above the opening of the eustachian tube. The curved end of the wall of this semicanal is called the cochleariform process. The lateral semicircular canal forms a broad bulging eminence in the floor of the passage between the tympanum and the antrum known as the prominence of the lateral semicircular canal. The entire cavity is lined with a mucous membrane whose surface is cuboidal, nonciliated epithelium. The mucosa is continuous with the membrane of the eustachian tube, and through it, with the mucosa of the nasal pharynx.

mastoid region The mastoid process is recognizable as a bony projection toward the end of the second year. As it gradually develops after birth it becomes pneumatized by a continuation of air spaces from the antrum. These *mastoid air cells* usually become larger toward the tip of the process, where one or more large spaces ordinarily occur in the adult. However, all the air cells may be small, or they may be absent owing to failure of pneumatization. The antrum and the mastoid cells are lined with a continuation of the membrane of the tympanic cavity. Pneumatization is a continuation of the developmental process. Formation of air cells is brought about by direct extension of epithelial

huds into newly forming bone or into older bone with bone marrow, but not into the bone marrow. When pneumatization occurs in older bone, the marrow cells first disappear and are replaced by loose connective tissue before invasion by the epithelium. In addition to the usual pneumatization of the mastoid process, the air cells may extend into the petrous pyramid, even reaching its apex in the wall of the middle cranial fossa. These cells, when they occur as outgrowths of the posteromedial wall of the tympanic cavity near the orifice of the auditory tube, are separated from the epitympanic cells by the solid bone of the otic capsule. Pneumatization, beginning in the same region,

Inflammation in the fetal middle ear from the presence of meconium or of vernix caseosa, or infection in postnatal life, may retard pneumatization with resulting diploic bone without air cells (Wittmaack). Acute inflammation of the tympanic cavity or antrum in early infancy may cause scarring and stop further growth of epithelial huds with a resulting diploic mastoid process without air cells. If it occurs in later infancy or childhood a partly pneumatic and partly diploic mastoid may result. Sclerotic bone in the mastoid is not produced by obliteration of previously existing mastoid cells from scar tissue or new bone formation due to inflammation, according to these views.

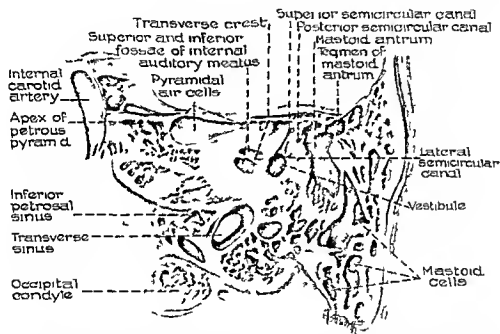


Fig. 193.—Section through mastoid process, mastoid antrum, vestibule, superior and inferior fossae of internal auditory meatus, and pyramidal part of temporal bone showing air cells.

may extend upward and backward between the arch of the carotid canal and the cochlea. It may also extend along this canal and downward and beneath it, producing an almost continuous circle of air spaces. Air cells may also form in the bony part of the auditory tube, in the floor of the tympanic cavity, and in the base of the zygoma. A large cell sometimes occurs internal to the digastric groove. Air cells in the lateral part of the petrous pyramid are inward extensions of epitympanic cells. Pneumatization varies in different subjects, with the possibility of different degrees of infection from infected mastoid cells.

In addition to the spina suprameatum, at the upper posterior margin of the external meatus the external surface of the mastoid is marked by the linea temporalis which forms a ridge at the upper boundary of the process. Remains of the petrosquamosal suture may also be present on the external surface in the adult. The completely pneumatized mastoid is large and more or less rounded. With incomplete pneumatization the process is smaller and the lateral sinus usually lies near the surface and near the posterior wall of the external meatus.

The anatomic relations of the mastoid process and its cells are of importance. The lateral

sinus lies along the inner wall at a varying distance from the outer wall of the mastoid and from the external meatus. The facial nerve enters the temporal bone with the acoustic nerve through the internal acoustic meatus and reaches the tympanic cavity above and in front of the oval window. It passes horizontally above the oval window to the posterior wall of the cavity, then turns downward and passes along the posterior margin of the cavity in the wall of the external meatus. Opposite the oval window it lies at a level with the inner wall of the tympanum. Passing downward toward the sty-

and a lateral or short process. The incus has a body, a short process lying in the incudal recess of the attic, and a long process which extends into the tympanic cavity. The stapes has a small head, a neck, and two arching processes, attached to the two ends of the base. The base is set into the oval window in such manner that its lower, posterior pole forms a fulcrum for rotation of the ossicle. The manubrium and the lateral process of the malleus are embedded in the tympanic membrane. The line of attachment of the manubrium, visible on the ear drum through the external meatus, forms an

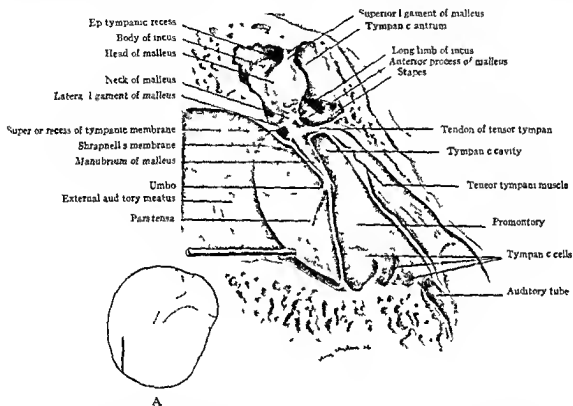


Fig 194—Frontal section through tympanic cavity and tympanic membrane A, Position of incision (From Callander, C L. *Surgical Anatomy*, Philadelphia, W B Saunders Company)

lomastoid foramen, it lies in the posterior wall of the external meatus and becomes gradually separated from the inner wall of the tympanic cavity.

The *auditory ossicles* are three small bones—the malleus, the incus, and the stapes—articulated together by small ligaments and attached to the walls of the tympanic cavity. They form a chain from the tympanic membrane to the oval window. They are covered with a thin mucous membrane, continuous with that of the tympanic cavity. The malleus consists of a head, neck, manubrium, an anterior or long process,

important landmark in otoscopic examination.

The chain of ossicles, with their articulations, forms a series of levers by which the movements of the tympanic membrane are transmitted through the base of the stapes to the perilymph of the labyrinth. The effective excursion of the stapedial base is reduced in about the ratio of 2 to 1, as compared with the center of the ear drum (Stevens, Davis, and Lurie), but its force is increased.

The *tensor tympani muscle* is about 22 mm long. It arises from the cartilaginous part of the

auditory tube, from the neighboring part of the great wing of the sphenoid, and from the walls of the semicanal in which it lies. It is attached to the manubrium of the malleus, near the neck, by a rounded tendon which passes over the cochleariform process almost at right angles, and then crosses laterally across the tympanic cavity. By drawing the manubrium medially this muscle tenses the tympanic membrane. It is innervated by the tensor tympani nerve, a branch of the mandibular division of the trigeminal, which reaches the muscle by way of the otic ganglion.

The *stapedius muscle* has its origin in the hollow of the pyramidal eminence. Its tendon emerges through the apex of this eminence and bends downward to be inserted into the posterior surface of the neck of the stapes. This muscle pulls the ventral border of the base of the stapes laterally. It receives its motor innervation by a twig from the facial nerve.

The Eustachian Tube—The auditory or eustachian tube extends from the anterior wall of the tympanic cavity to the nasopharynx. It has a length of about 38 mm and its direction is downward, forward, and medialward. The posterior third is surrounded by the temporal bone and lies anterolateral to the carotid canal. The anterior two thirds is partly cartilaginous and partly fibrous. It lies in a narrow groove between the greater wing of the sphenoid and the petrous portion of the temporal bone. The tensor palati muscle separates it laterally from the mandibular nerve and the middle meningeal artery.

The tube flares out as it opens through the lateral wall of the nasopharynx, passing over the upper border of the superior constrictor muscle at the posterior border of the medial pterygoid plate. The posterior upper margin, known as the torus tubarius, forms a prominent fold. The tympanic opening of the tube also flares out. At the junction of the bony and cartilaginous parts, the lumen is narrowed into the isthmus, which is flattened and measures 1 to 2 mm in diameter. The cartilaginous portion is supported by a grooved plate of hyaline cartilage, the upper part of which curves laterally and downward over the tube. The greater part of the lateral wall, however, is formed of a membrane of connective tissue. The mucous membrane of the bony portion has a low columnar, ciliated epithelium. Near the nasopharynx, tall, pseudostratified ciliated epithelium

occurs, with numerous goblet cells near the pharyngeal orifice.

The tympanic orifice of the auditory tube is too high to provide effective drainage of the tympanic cavity. In children the tube is shorter, broader, and more horizontal than in adults, and provides better drainage. However, ascending infections from the pharynx also are facilitated. The narrow isthmus is easily closed by swelling of the mucous membrane, and longstanding infection may result in permanent constriction due to scar formation. Surrounding the pharyngeal orifice there is a ring of lymphoid tissue in the mucosa, sometimes called the tubal tonsil of Gerlach.

The Inner Ear—The inner ear or labyrinth comprises a series of cavities and canals in the petrous portion of the temporal bone. The bony canals and spaces constitute the bony labyrinth. They are lined with perosteum and enclose the membranous labyrinth.

The Bony Labyrinth—The bony labyrinth is surrounded by a capsule of bone 2 to 3 mm thick, embedded in the pyramid of the petrous part of the temporal bone. The capsule consists of three layers, namely, an outer perosteal layer, a middle layer derived from the cartilage surrounding the embryonic membranous labyrinth, and a thin inner layer derived from the endosteum of the bony cavities. The inner layer has no blood vessels and is of ivory like hardness. The adjacent side of the middle layer is also dense, but the external portion next to the outer layer is of looser texture. The middle layer often is characterized by remnants of cartilage. In otosclerosis the changes from dense bone into a loose, spongy bone begin in this layer. The cavities and canals of the bony labyrinth are the vestibule, the three semicircular canals and their ampullae, the vestibular aqueduct, and the cochlea.

The *vestibule*, situated medial to the tympanic cavity, is an irregular oval chamber about 4 mm in diameter. Its lateral wall is perforated by the oval and round windows. Anterosuperiorly the vestibule leads into the cochlea, and postero-inferiorly the ends of the semicircular canals open into it. On the superior and posterior part of the medial wall the spherical recess contains the saccule. Between the two is found the vestibular crest which divides posteriorly into two limbs that bound a small concavity, the cochlear recess, at which the cochlear duct begins. The posterior wall has the opening of

the vestibular aqueduct which transmits the endolymphatic duct and a small vein

There are three *semicircular canals*. Each forms about two thirds of a circle and has a diameter of about 1 mm, save at one end where each dilates into an *ampulla*, about 2 mm in diameter. The *superior canal*, 15 to 20 mm long, and the *posterior canal*, 18 to 22 mm long lie in nearly vertical planes at right angles to each other, the angle opening laterally. The non-ampullated ends join to form a common *crus* which opens on the medial wall of the vestibule. The anterolateral end of the superior canal and the inferior end of the posterior canal have ampullae which open into the superior and in-

terior parts of the vestibule, respectively. The *lateral semicircular canal*, 12 to 15 mm long, is the shortest of the three and bends outward in the horizontal plane. Its ampulla opens into the upper part of the vestibule above the oval window near the orifice of the superior canal. The superior canal lies in a plane at right angles to the long axis of the petrous bone, the plane of the posterior canal is parallel to the long axis of the bone, and the lateral canal is placed in a horizontal plane. The superior canal of one side is parallel to the posterior canal of the other side, while the lateral canals of the two sides occupy the same plane.

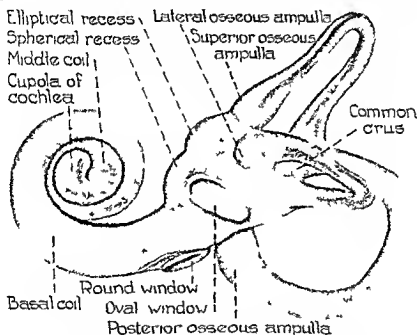


Fig. 195.—Wood's metal cast of human labyrinth, anterior view.

terior parts of the vestibule, respectively. The *lateral semicircular canal*, 12 to 15 mm long, is the shortest of the three and bends outward in the horizontal plane. Its ampulla opens into the upper part of the vestibule above the oval window near the orifice of the superior canal. The superior canal lies in a plane at right angles to the long axis of the petrous bone, the plane of the posterior canal is parallel to the long axis of the bone, and the lateral canal is placed in a horizontal plane. The superior canal of one side is parallel to the posterior canal of the other side, while the lateral canals of the two sides occupy the same plane.

The *cochlea* is a flattened cone 9 mm in di-

ameter at the base and 5 mm high. It consists of a central column, the *modiolus*, around which a hollow spiral tube makes two and one half to two and three quarters turns. The tube is about 32 mm long. The cochlear apex is directed forward and laterally, the base backward and medially. The base forms part of the floor of the internal auditory meatus. The tube is divided into an *anterior scala vestibuli* and a *posterior scala tympani*, between which lies the *scala media* or *cochlear duct*. In most figures of the cochlea its long axis is shown as vertical, in which position the *scala vestibuli* lies above the *scala tympani*. The two *scalae*, which taper irregularly toward the apex, become continuous

under the cupola of the cochlea, being joined by a narrow canal, the *helicotrema*.

The *modiolus* forms a conical central core of the cochlea. It has a spirally arranged series of small foramina through which the cochlear nerve and its twigs reach their terminations in the organ of Corti. These are the longitudinal *modiolar canals* and the *spiral modiolar canals*. They also transmit blood vessels to the cochlea. A shelf of bone, the *osseous spiral lamina*, projects into the cochlear canal, partly dividing the *scala vestibuli* from the *scala tympani*. The partition is completed by the *basilar membrane*. The apical end of the spiral lamina is known as the *hamulus*. A canal which winds around

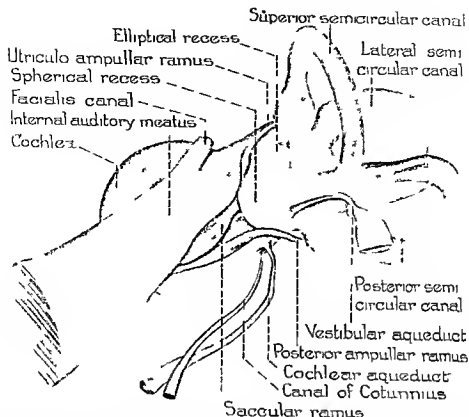


Fig. 196—Posterior view of Wood's metal cast of bony labyrinth, showing the internal auditory meatus, the channels for vestibular and cochlear nerves, the vestibular and cochlear aqueducts and the canal of Cotunnus for transmission of the vein of the cochlear aqueduct

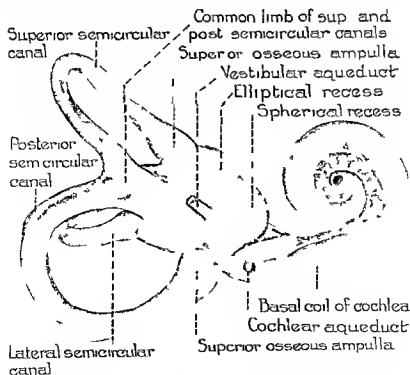


Fig. 197—Posterior view of Wood's metal cast of right bony labyrinth

the modiolus at the base of the spiral lamina contains the spiral ganglion. The basal part of the bony cochlea has three openings, namely, the opening into the vestibule, the round window, and the opening of the cochlear canaliculus (perilymphatic duct) through which a connection is made with the subarachnoid space.

The Membranous Labyrinth—The membranous labyrinth is much smaller than the spaces it occupies in the bony labyrinth. The perilymphatic space, between it and the periosteum of the surrounding bone, is filled with perilymph. Fibrous strands connect the membranous labyrinth with the periosteum, producing irregular

saccular duct, the two parts of which unite to form the endolymphatic duct, a slender membranous tube which passes through the vestibular aqueduct and ends under the dura of the posterior surface of the temporal bone as the endolymphatic sac. At the opening of the utricle into the utricular branch of the endolymphatic duct is found a valve like structure, the utriculo-endolymphatic valve (Bast), directed toward the utricle. The sacculus is connected with the membranous labyrinth of the cochlea through the ductus reuniens.

The *utricle* is an ovoid, somewhat flattened sac whose rounded end is firmly attached to

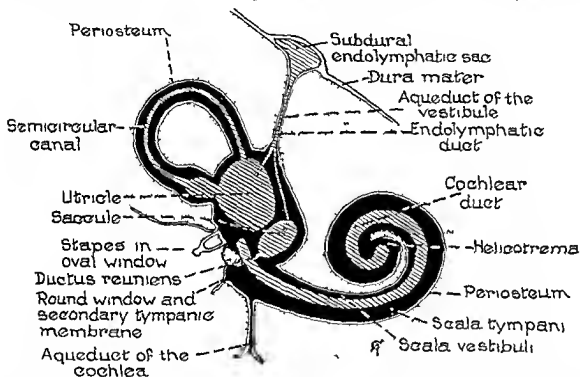


Fig. 198.—Diagram of perilymphatic and endolymphatic spaces of the internal ear (From Larsell, O. *Anatomy of the Nervous System*, New York, D. Appleton Century Company.)

spaces comparable to the subarachnoid cavity surrounding the central nervous system. The membranous labyrinth is filled with endolymph. It includes the neuro epithelial patches and the organ of Corti which together form the vestibular and the cochlear receptors. The latter are innervated by the cochlear nerve, whose spiral ganglion (of Corti) lies in the modiolus. The vestibular sensory cells are supplied by the vestibular nerve, the vestibular ganglion (of Scarpa) lying at the bottom of the internal auditory meatus.

Two enlargements of the membranous labyrinth, the utricle and the saccule, occupy the vestibule. They are connected by the utriculo-

saccular duct, the two parts of which unite to form the endolymphatic duct, a slender membranous tube which passes through the vestibular aqueduct and ends under the dura of the posterior surface of the temporal bone as the endolymphatic sac. At the opening of the utricle into the utricular branch of the endolymphatic duct is found a valve like structure, the utriculo-endolymphatic valve (Bast), directed toward the utricle. The sacculus is connected with the membranous labyrinth of the cochlea through the ductus reuniens. The *utricle* is an ovoid, somewhat flattened sac whose rounded end is firmly attached to

hairs of the hair cells penetrate. The upper part of the membrane contains small prisms, known as otoconia or otoliths, composed of calcium carbonate and a protein.

The *sacculus* is nearly spherical and is smaller than the utricle. It lies in front of the utricle in the anterior and inferior part of the vestibule. On its medial wall the macula sacculi forms a heart shaped patch of sensory epithelium, about 2 by 3 mm in size, corresponding to the macula of the utricle. The surface of the saccular mac-

external transverse groove on its attached surface through which the ampullary branch of the vestibular nerve enters. Internally and overlying the groove there is a ridge of neuro-epithelium, the ampullary crest, which projects into the lumen of the duct. The fibers of the ampullary twigs of the vestibular nerve end about hair cells in this crest. The crista is covered by a caplike gelatinous mass, the cupula, into which the hairs of the hair cells penetrate. Movements of the endolymph from the canal

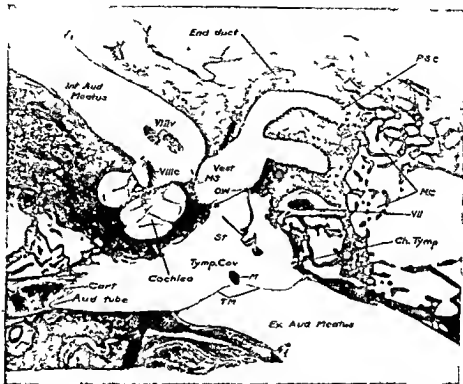


Fig 199—Section through left temporal bone of man (age seventeen years). *Aud tube*, Auditory (eustachian) tube; *Cart*, cartilage of auditory tube; *Ch tym*, chorda tympani nerve; *End duct*, endolymphatic duct; *Ext Aud Meatus*, external auditory meatus; *Int Aud Meatus*, internal auditory meatus; *M*, manubrium of malleus; *MC*, mastoid air cells; *MS*, macula sacculi; *OW*, oval window; *St*, stapes; *TM*, tympanic membrane; *Tym Cav*, tympanic cavity; *Vest*, vestibule; *VII*, seventh cranial (facial) nerve; *VIIIc*, cochlear division of eighth cranial (auditory) nerve; *VIIIv*, vestibular division of eighth cranial nerve. (Photomicrograph of a section from the collection of Dr E. P. Fowler Jr. in *Bailey's Histology*, 10th ed., edited by Philip E. Smith. Baltimore: The Williams and Wilkins Company.)

ula, however, follows the sagittal plane in part and is thus perpendicular to the surface of the macula utriculi. It is also covered by an otolithic membrane. Its hair cells are surrounded by terminal twigs of the saccular branch of the vestibular nerve.

The *semicircular ducts* are membranous tubes within the bony semicircular canals, about one fourth the diameter of the latter. In the ampullae of the canals the membranous ducts also are ampullated. Each membranous ampulla has an

to the utricle, or vice versa, exert pressure on the cupula and through it stimulate the hair cells of the crista.

The membranous part of the cochlea is the *cochlear duct* or *scala media*. For the sake of simplicity its parts will be described with reference to the cochlea when the longitudinal axis of the latter is in the vertical position. The cochlear duct begins in the cochlear recess of the vestibule as a blind pouch, the vestibular sacculus. Following the spiral canal of the bony

cochlea as a tube, triangular in cross-section, it forms a basal, a middle, and an incomplete apical turn, and ends blindly just beyond the hamulus as the cupular cecum. Its floor consists of the basilar membrane and the limbus spiralis. The latter is a ridge-like elevation formed by the periosteum of the upper surface of the spiral lamina, with a covering of epithelium. The highly specialized structures of the organ of Corti and its related cells rest on the basilar membrane and project upward into the duct. Lateral to the organ of Corti the ex-

tutes the stria vascularis, containing numerous blood vessels and capillary loops which extend into its covering epithelium. It is usually regarded as the source of the endolymph, but Shambaugh has demonstrated glandlike structures in the wall of the external spiral sulcus. The roof of the duct is formed by the vestibular or Reissner's membrane. This is but 3 microns thick and is attached internally near the free margin of the bony spiral lamina and peripherally to the spiral ligament, forming an angle of about 45 degrees with the basilar membrane.

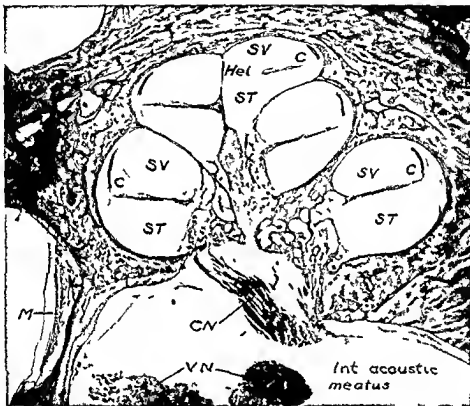


Fig. 200—Axial section of cochlea and edge of sacculus from fifteen year old girl. C, Cochlear duct, CN, cochlear division of auditory nerve, Hel, helicotrema, M, macula sacculi, ST, scala tympani, SV, scala vestibuli (Photomicrograph of a section from the collection of Dr. E. P. Fowler, Jr. in Bailey's *Histology*, 10th edition, edited by Philip E. Smith, Baltimore, The Williams and Wilkins Company.)

ternal spiral sulcus forms a trough whose outer boundary is the spiral prominence. The internal spiral sulcus forms a deep groove between the spiral limbus and the axial attachment of the basilar membrane. The upper margin of this groove is known as the vestibular lip and the lower margin as the tympanic lip. The peripheral wall of the duct is formed by thickened periosteum of the outer wall of the bony canal known as the spiral ligament of the cochlea, covered with irregular, pseudostratified epithelium. The upper part of this ligament consti-

tutes the stria vascularis, containing numerous blood vessels and capillary loops which extend into its covering epithelium. It is usually regarded as the source of the endolymph, but Shambaugh regards as representing a lamellar structure. The membrane has a planoconvex form in section. It is narrow and thin in the lower part of the basal coil, but gradually widens and thickens toward the apex of the cochlea. Most methods of preparation for histological study distort and detach it so that the membrane appears to float in the endolymph,

anchored only at the vestibular lip. Along its lower surface, opposite the row of inner hair cells, a dark stripe of Hensen represents a facet of attachment to the tall border cells medial to the inner hair cells. The lower surface is also attached (as Shambaugh, Prentiss, and others have shown) to the hairs of the hair cells. The number of points of attachment, using the mean of the number of hairs on inner and outer hair cells, as given below, would approximate 2,000,000—an average of about 62,500 per mm of length. However, the middle and apical coils of the cochlea, where there are four to five

coil it thickens and in some animals may rest on bone. It is divided into an inner zona arcuata, extending from the tympanic lip to the bases of the external pillars, and a zona pectinata, from these pillars to its attachment to the spiral ligament. It consists of a vestibular covering layer, a middle layer, made up of basilar fibers embedded in a homogeneous ground substance, and a tympanic covering layer. The basilar fibers, numbering about 24,000, range in length from 64 to 128 microns at the beginning of the basal coil, and 352 to 480 microns near the apical end of the mem-

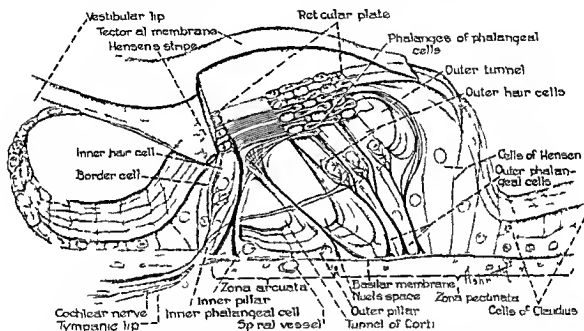


Fig 201—Semidiagrammatic representation of the organ of Corti and the tectorial membrane, based on the interpretation that the latter is attached along Hensen's stripe and at its outer margin. A portion of the tectorial membrane is represented as cut away to show more clearly the reticular plate phalanges and ends of the hair cells. The remainder is depicted as translucent. The pillars of Corti and the outer phalangeal cells show the arrangement of the tonofibrils (black lines) and their manner of spreading out in the head processes of the pillars, in the phalanges, and below the basal ends of the hair cells. (Adapted from Retzius, Held, Testut, Shambaugh, and others.)

rows of outer hair cells, have a larger number of attachments per millimeter of length than the basal coil, with but three rows of hair cells. Some histologists hold that the outer margin of the membrane is attached to the cells of Hensen.

The basilar membrane extends from the free border of the tip of the spiral lamina to the crest of the spiral ligament. Its breadth increases from 80 microns at its basal end to 498 microns about one half turn from its apical termination, toward which it then narrows rapidly (Wever). At the lower end of the basal

brane. In the zona pectinata the fibers are straight, smooth, and unbranched, with a diameter of 1 to 2 microns. In the zona arcuata they are thin, branching elements arranged as a network. The tympanic covering layer is made up of delicate connective tissue containing blood capillaries in the inner zone. A spiral vessel is usually present under the zona arcuata. The zona pectinata has no blood vessels.

The organ of Corti or papilla spiralis is the auditory receptor apparatus. It consists of a framework of highly specialized epithelial supporting cells and a series of neuro-epithelial

elements, the hair cells. Above the organ, as usually described, lies the tectorial membrane. In the normal position of the cochlea, the base of the organ of Corti lies in a nearly vertical plane and the broad axis of the tectorial membrane is nearly parallel to it, lying rostrally to, not above, the papilla spiralis.

The supporting apparatus of the organ of Corti is complex and elaborate in structure. Its central feature is the tunnel of Corti, bounded by the inner and the outer pillars or rods of Corti. Near the basal end the inner and outer pillars are about 50 microns long. They gradually increase in length, the outer ones more rapidly, so that near the helicotrema they measure 85 to 100 microns, respectively. The tunnel widens from 20 microns at the base to 85 microns near the apex.

The pillars rest on the basilar membrane. The heads of the two series are joined in an articulation-like structure and continue laterally as flattened processes which merge with the reticular plate. The outer phalangeal cells (cells of Deiters) also rest on the basilar membrane. The inner side of each has a shoulder which supports the adjacent hair cell. A tapering and curving process continues between the rows of hair cells to the reticular plate, where it forms a phalange. The phalanges are offset apically from the bases of their cells by the width of two to three hair cells. Three, four, and five rows of Deiters's cells occur in the basal, middle, and apical coils, respectively. Their phalanges interlock to form the main part of the reticular plate which has perforations for the ends of the hair cells. Bundles of tonofibrils in the cells give off branches to the shoulders and long, slender bundles to the phalanges. They divide into individual fibrils, forming rays in the phalanges and calyces in the shoulders. The spaces of Nuel and the outer tunnel are continuous with the tunnel of Corti through clefts between the pillars. The cells of Hensen, resting on the basilar membrane lateral to the Deiters's cells, form the outer support of the reticular plate and probably are the attachment for the margin of the tectorial membrane. Laterally they are continuous with the cells of Claudius, beneath which, in the basal coil, lie the cells of Boettcher.

The inner phalangeal cells lie against the inner surface of the inner part of the reticular plate. The cuticle of the border cells, which lie medial to the inner hair cells, forms the inner

boundary of the openings for the latter and attaches to the inner phalanges, constituting the margin of the reticular plate.

The structure of the entire supporting apparatus suggests a resilient cushion in which the tonofibrils of the pillars and phalangeal cells serve as minute springs. This conception is strengthened by the articulation-like arrangement of the heads of the pillars, whose slender processes attach to the reticular plate, and by the offset of the processes of the phalangeal cells before the latter also become part of the reticular plate. The inner part of the plate, lying above the pillars, would appear less resilient than the outer portion.

THE HAIR CELLS—The special sensory cells of the organ of Corti are columnar elements with a rounded base and a number of stiff hairs at the opposite end. The cytoplasm is finely granular and stains feebly. In the lower part of the cells the granules are larger and stain more deeply. These cells convert the refined mechanical stimulation received through their hairs into nerve impulses, transmitted by the fibers of the cochlear nerve. Stimulation probably is effected by pressure or pulling of the hairs through interaction of the basilar membrane, the supporting framework of the organ of Corti, and the tectorial membrane.

The inner hair cells, numbering about 3500, form a single spiral row wedged between the tall inner border cells and the inner pillars of Corti. The bodies of the cells, about 12 microns in diameter, rest obliquely against the inner pillars of Corti. The distal ends project into openings in the reticular plate and have forty-one to sixty-four hairs arranged in parallel rows.

The outer hair cells form three rows in the basal coil, four in the middle coil, and five in the distal part of the cochlea. They number about 20,000 and have a diameter of 8 microns. The head of each cell extends into an opening in the reticular plate through which its hairs, numbering eighty-three to one hundred, protrude. The rounded base rests on the shoulder of the laterally adjacent Deiters's cell, above described.

THE COCHLEAR NERVE AND SPIRAL GANGLION—The organ of Corti is innervated by the cochlear nerve which arises from bipolar ganglion cells of the spiral ganglion. In ten young adults these cells numbered 23,193 to 39,114 with an average of 29,024 (Guild). The ganglion cells give off peripheral processes which form a

spiral mass of bundles that gradually fray out into small groups of fibers to the organ of Corti. The fibers lose their myelin sheaths at the *foramina nervosa* at the tympanic lip and pass to the hair cells. Some end on the inner hair cells as small buttons on terminal twigs, while others cross the tunnel of Corti to terminate in the same manner about the outer hair cells.

According to Lorente de No, there are radial fibers and spiral external fibers. The radial fibers pass directly to the inner hair cells, each giving terminal twigs to only a few cells, but twigs from several fibers may end on the same hair cell. The spiral external fibers bend at a right angle toward the base of the cochlea when they have reached the radial level of the outer hair cells and continue one third of a cochlear turn or more, giving off twigs to numerous outer hair cells. The central processes of both types collect in the modiolus to form the main bundle of the cochlear nerve. On reaching the medulla oblongata each fiber divides into an ascending branch, to the ventral cochlear nucleus, and a descending branch to the *tuberculum acusticum*.

The Blood Supply of the Labyrinth—The labyrinth is supplied by the labyrinthine artery which may stem from the basilar or the inferior cerebellar artery. In the internal acoustic meatus, it gives off a cochleovestibular branch, which divides into a trunk supplying the proximal two thirds of the basal coil of the cochlea, and a posterior vestibular artery, supplying the macula sacculi, the ampulla of the posterior canal, the common crus, and the posterior crura of the posterior and lateral canals, in addition to the periosteum of the vestibule. A continuation of the labyrinthine artery divides into two trunks, one entering the modiolus and supplying all of the cochlea save the proximal two-thirds of the basal coil, and the other, the anterior vestibular artery, supplying the macula acustica utriculi and the cristae and anterior crura of the superior and lateral canals. These branches are all terminal arteries, constituting the sole blood supply for their respective areas of distribution. The venous drainage of all of the cochlea and part of the vestibule and semicircular canals passes through a small vein accompanying the cochlear aqueduct, while the remainder of the labyrinth is drained by a vein emerging through the vestibular aqueduct.

Stimulation of Hair Cells—It is generally accepted that the vestibular hair cells are stimu-

lated by slight displacements of their overlying cupulae or otolithic membranes. In the cochlea, however, the complexity of structure makes the method of stimulation difficult to analyze. It is generally accepted that the cochlea behaves as if it contains a series of resonators affected by tonal vibrations of different rates. According to the hitherto more generally held view of the resonance theory, stimulation is brought about by vibrations of the basilar membrane set up by waves in the perilymph due to movements of the stapes at the oval window. Helmholtz regarded the basilar fibers as resonators affected by specific tones. However, much of the elaborate anatomic structure is inadequately explained by this theory, while there are difficulties in the way of accepting the basilar membrane as a resonance vibrator. The vibration amplitudes of the tympanic membrane necessary to produce audible tones (Wilska) are so minute at ordinary tones and volumes of sound, that the movements of the basilar membrane must be infinitesimal (Stevens, Davis, and Lurie). If the tectorial membrane is closely adherent to the organ of Corti, as good histologic evidence indicates, and if the supporting framework of the latter is a resilient cushioning apparatus between the two membranes, as its structure suggests, the difficulties of considering the basilar membrane as a resonator are increased.

Localized receptivity in the organ of Corti, with high tones in the basal coil and successively lower tones toward the apex, has been demonstrated by clinicopathologic as well as experimental studies. Recent studies indicate that with increase in intensity the response spreads to adjacent areas, more so for low than for high tones. For successive octaves the zones of optimal response occur at equal distances along the organ of Corti (Walzl and Bordley).

Evidence that the pillars of Corti and the remainder of the supporting apparatus serve as structures necessary to hearing in mammals is adduced from the relation of the differentiation of this apparatus and the related tunnel to the inception of hearing in developing opossums. The tectorial and the basilar membranes both appear to be well differentiated long before hearing appears, as also is true of the hair cells and their nerve fibers. The pillars of Corti and the remainder of the supporting apparatus undergo a slower differentiation which however,

must reach an approximately adult stage before function begins at the various levels of the cochlea. It therefore appears reasonable to assume that this apparatus plays some part other than merely holding the hair cells in place. If it be regarded as a resilient apparatus between the two membranes, an appropriate tension between them would be possible, favoring stimulation of the hair cells at optimal points by even slight movements or pressures. As it completes its differentiation it also increasingly refines the receptor mechanism as shown by increased sensitivity of the opossum cochlea in later stages of the pouch young (Larsell, McCrady, and Larsell).

The basilar membrane shows an elongated wave of vibration which flattens out toward the helicotrema (Bekesy). Reboul has calculated vibration waves of similar form in fluid contained in a conical tube with an elastic wall. In addition, he derives pressure waves of different form and phase. The lengths of both vary with the vibration rate producing them.

For a review of the numerous theories attempting to correlate structure and function of the cochlea, the reader is referred to the excellent historical survey by Bast and Shover. In many of the recent experimental studies of the cochlea, the behaviour of the basilar membrane has been emphasized almost to the exclusion of other structures, save the hair cells. The cochlea is structurally the most intricate organ of the body, refined for the reception of minute mechanical stimuli, and presumably all parts have their functions. Considering the tectorial and basilar membranes, the supporting framework of the organ of Corti and the hair cells as a closely knit unit which functions by the interaction of all its parts, some such physical force as Reboul's waves, transmitted through the perilymph and endolymph, and possibly modified by the latter, may account for a larger number of factors than any mode of hair-cell stimulation hitherto proposed.

Central Connections of Membranous Labyrinth.—VESTIBULAR CONNECTIONS.—The central processes of the vestibular nerve terminate directly in the vestibular nuclei and in the flocculonodular lobe of the cerebellum. Here secondary connections are made which govern the equilibrium of the body. Spiegel has adduced evidence of a cortical connection also.

COCHLEAR CONNECTIONS.—The cochlear fibers enter the medulla oblongata and divide

into ascending branches that pass to the ventral cochlear nucleus, and descending branches to the tuberculum acusticum. The division occurs along an arc at the caudal boundary of the ventral nucleus that is a projection of the uncoiled ganglion of Corti, the highest point of the arc corresponding to the apical and the lowest point to the basilar part of the ganglion (Lorente de No).

The thalamic projection of the auditory path in the medial geniculate body is curvilinear, representing a point-to-point projection from the cochlea (Ades, Mettler, and Culler). The auditory cortex of the temporal gyri likewise shows specific areas for reception of various tone ranges (Woolsey and Walzl, in the cat, Tunturi, in the dog). There thus is established a point-to-point connection between the cochlea and the principal central auditory centers.

OLOF LARSELL

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PHYSIOLOGY OF THE EAR

Physiology of Hearing.—Perception of sound waves, alternate condensation and rarefaction of air, occurring at rates from 16 to 22,000 double vibrations (cycles) per second, constitutes "hearing" for the human species. Sounds below 16 cycles may be felt as motion, while those above 11,000 are perceived by few. Our useful conversational range lies between 256 and 2048 cycles. Behavior of certain animals indicates sensitivity to vibrations reaching 40,000 to 50,000, so-called "suprasonic" vibrations measurable by physical means.

Sounds as perceived by the human ear are rarely pure tones. Human voices, the notes of various musical instruments, and the cries of animals are composed of dominant tones modified by harmonic overtones. Thus are preserved the characteristics by which the note of a violin differs from that of a clarinet, or by which we identify the voices of friends. Each dominant tone and its overtones must attain simultaneous reception and identification by both internal ears, and simultaneous interpretation by both sides of the auditory cerebral cortex. Noises, similarly perceived, are composed of mingled tones lacking harmonic congruity. Excessive loudness or repetition both of harmonious sounds and of noises may, through excessive vibratile stimulation, lead to congestion, inflammation, and eventual permanent damage both to transmitting and receptor mechanisms in the ear.

Sound waves pass into the external ear and its doubly bent canal and may be impeded there by structural narrowing, exostoses, or accumulations. Reaching the tympanic membrane, sound waves are transformed into lateral motion, which is communicated through the handle and body of the malleus and its solid arthrodesis with the body of the incus, to the head of

the stapes. The ossicular chain swings on an anteroposterior axis of ligamentous structures about the necks of the malleus and incus, and increases the intensity of each sound impulse more than two fold, while correspondingly diminishing its amplitude of movement. Sudden or severe shocks to the ossicular apparatus are further guarded against by the tensor tympani muscle, running forward from the neck of the malleus, and by the stapedius muscle, attached to the neck of the stapes and pulling its foot plate outward. Tightening these muscles lowers the threshold of sound reception by 10 to 30 decibels. Sound more intense than 90 to 100 decibels usually is painful.

Sound may also reach the cochlea by vibrations conducted through the bones of the head, but much greater intensity is required for bone conduction than for air conduction. Moreover, when the tympanic membrane has been lost, excellent hearing by air may be present, although intensity must be somewhat greater in order that the ossicles, especially the stapes, may pick up adequate stimuli. If the tympanic membrane is absent and the ossicular chain more or less destroyed, with the stapes embedded in scar tissue, the round window may receive maximum stimulation. This accounts for the good results secured by cellophane-bristle hearing prostheses (Pohlmán) and by oil-moistened cotton balls against the promontory.

Ossicular motion in the air filled middle ear may be limited by reduction of the external air pressure, when failure of the eustachian tube to open may cause increased intratympanic pressure and bulging of the tympanic membrane or, by increased external air pressure, with diminished intratympanic pressure and inward drawing of the tympanic membrane. Proper functioning of the eustachian tube, opening and closing during swallowing, is thus a condition of adequate hearing.

Lateral motion of the ossicular chain reaching the stapes causes its footplate to pivot gently on its thickened lower margin, as it stands sealed into the oval window by a firm membrane. Thus the intensified motion of the ossicles is transmuted into a succession of fluid waves, proceeding according to the laws of hydrodynamics into the endolymph contained in the scala vestibuli of the cochlea (Fig 202). These fluid wave impulses are conducted along the scala vestibuli the entire length of the cochlea to its apex, the helicotrema, where they cross

over and follow the reverse course down the scala tympani, to beat upon the membrane of the round window, which is looked upon by some as a possible equalizing or decompressing device against sudden or excessive intracochlear pressures. The ductus reuniens, proceeding backward to the sacculi and thus connecting with the semicircular canals, may share this function of decompression against noise.

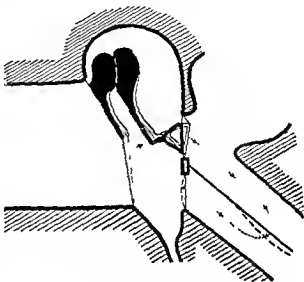


Fig 202 — Schematic diagram of the tympanic membrane, the ossicles, and the basilar membrane. The solid figures of the ossicles and the solid lines for the tympanic, basilar, and round-window membranes are in their resting positions. The open outlines of the ossicles and broken lines for the membranes show their positions following inward displacement of the tympanic membrane by a sound wave. (Reprinted by permission from "Hearing: Its Psychology and Physiology" by Stevens and Davis, published by John Wiley & Sons, Inc.)

Localization of perception of high tones in the basal cochlear coil near the oval window, with the lowest tones crowded close together near the helicotrema, has been thoroughly established by Crowe, Guild, and Polvogt, and by Culler. The nerve endings of the organ of Corti, which lies upon the basilar membrane and projects upward into the ductus cochlearis, are acted upon by fluid waves carried by the endolymph of the scala vestibuli. The dimensions of these membranous canals are minute, and some 25,000 to 29,000 ganglion cells, each with numerous hair-cell connections in the organ of Corti, are crowded into the spiral cochlear ganglion. Obviously there is in this limited compass no such free propagation of sound waves as in an air-filled cavity, nor can the results from electric currents, picked up and

vastly amplified as they pass into the trunk of the auditory nerve, analyze and decipher the exact method by which the cochlear endings distinguish between various sounds with their identifying overtones

According to earlier theories, sounds either set in motion fibers in the basilar membrane which acted as "resonators," or these fibers were thought to act as microphonic "receivers," by virtue of the hair cells to which they are connected. Recent experimentation has inclined to the microphonic theory, especially since electric stimulation of cochlear areas has been found to produce characteristic oscillographic images for corresponding tones, in accurately mapped areas of the auditory cortex of both hemispheres. The most plausible recent theory, however, has been advanced by Reboul and is based on his accurate computations of the hydrodynamics involved in the propagation of fluid wave motion within such tiny, elongated, conical cavities as the scala vestibuli and the scala tympani. The length of pressure waves and waves of elongation set up by each tone is a function of the vibratory frequency of that tone, and such waves will impinge upon the receptor endings of the organ of Corti at specific points. This theory makes it unnecessary to assume that thousands of little microphones are lined up in the cochlea waiting for tones to locate them. Contrariwise, fluid vibrations at a given frequency will always produce sufficient pressure at a mathematically determinable point to activate a relatively simple perceptive mechanism. Much more proof of these propositions is, of course, needed. This theory has received experimental confirmation from Larsell's histologic studies of the developing cochleas of pouched opossum embryos, from which movement reflexes and oscillographic responses were secured only as various areas reached functional activity. One may therefore assume that differentiation of tone perception takes place in the cochlea.

Impulses from the cochlea travel up the auditory nerves and tracts and are distributed through the medial geniculate bodies to the auditory cortex of each cerebral hemisphere. A map of the cochlea may now be plotted in this area, this has been done, first in the cat, by Woolsey and Walzl, through serial electric stimulation of nerves entering the cochlea at different levels, and more recently in the dog, by Tunturi, who used pure tones as well as

direct electric stimulation of the cochlea, and the oscillographic tone images from the brain surface. It seems probable that understanding of the character and type of sound must be a function of the auditory cortex, as it sorts out the multiplex successive impulses arising from differentiation by the cochlear nerve endings.

Physiology of Equilibrium—The vestibular apparatus contributes a most important group of factors to human equilibrium, since its three interconnected semicircular canals on each side are paired in three planes of space, in such fashion that two will receive stimulation from each direction of motion of the head. Maintenance of steadiness during motion is "dynamic" equilibrium.

Each canal has a terminal ampullar widening in which numerous hair cells are grouped together into a plate (crista), projecting into the endolymph and capped by a thin gelatinous layer (cupula). Motion of fluid past these hair cells, perhaps swinging of the cupular weight upon them, sets up stimuli passing through the vestibular nerve to the central vestibular pathways, to the flocculonodular lobe of the cerebellum, and, through the medial longitudinal bundle, to the nuclei of the external eye muscles. Postural and muscular correction of these vestibular stimulations are brought about by cerebellar coordination and cortical reference to motor areas, with consequent reflex or voluntary response.

Excessive stimulation reacts upon the ocular muscle nuclei to produce vestibular nystagmus—fast movement away from the stimulated canal, with a slow recovery component pulling the eyes back into line.

The sacculi, which contain a similar gelatinous plaque with granules of lime salts (otoliths) lying upon its macula of hair cells, is connected anteriorly with the cochlea, and thus might serve to transmit sound from an opened bony semicircular canal. The utricle, similarly provided with otolithic endings, is believed to aid in nonmobile (static) equilibrium.

Equalization of fluid pressure within the labyrinth may be aided by posterior connection through the minute ductus endolymphaticus with its subdural saccus, on the cerebellar surface of the petrous bone.

Other factors intimately concerned with equilibrium, such as muscular, visceral, and tactile sensations, are related to the vestibular apparatus through its cerebellar connections, through

the vestibulospinal tracts, and overflow into the sympathetic system with corresponding disturbances in visceral organs

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AURAL AND NASAL NEURALGIAS AND NEUROSES

Aural Neuralgias—Aural neuralgias, aside from those due to local inflammatory changes, may be caused by such local physical factors as increased or diminished intratympanic pressure from sudden alterations in external air pressure—diminished, in rapid elevator or airplane ascent, increased during descent, and particularly increased in air locks during tunnel construction, caisson diving, and similar occupational hazards, or as a result of bomb blast or artillery fire

Pain referred to the ear may arrive by several routes (1) From the sphenopalatine ganglion, great superficial petrosal nerve, geniculate ganglion, sensory seventh to the posterior surface of

the external auditory meatus, concavity and back of the pinna, and mastoid area These pains may arise from sphenoiditis or ethmoiditis, pressure on the middle turbinate by high septal thickening or deviation, ulceration or neoplasm of the upper nasopharynx, or by retrolubular orbital tumor Such pain is also prodromal to and accompanies herpes zoster oticus (2) From glossopharyngeal, palatine, and superior laryngeal connections to the tympanic plexus, eustachian tube, and external auditory canal Such irritative phenomena as laryngeal and epiglottic tuberculosis and carcinoma, lymphosarcoma and the lymphoid enlargements of Hodgkin's disease and leukemia, ulcerative or purulent lesions of the pharynx, tonsils, and tongue, or of the upper esophagus, may be referred to the ear along this route (3) From the superior and inferior dental branches of the maxillary and mandibular divisions of the trigeminus, including branches to the temporomandibular articulation, and referred to the tympanic plexus Poor occlusion of teeth or plates (causing the mandible to slip forward), impaction of molars, apical abscess, pulp stones, epulis, acute parotitis, sublingual or submaxillary abscess or calculus, and incipient facial palsy may cause aural pain along this route

Petrosal inflammation or purulent accumulation arouses pain in the parietal region of the affected side, as well as abducens paralysis (Gradenigo's syndrome) Such pain is due to pressure forward against the dural envelope of the gasserian ganglion as it lies just anterior to the petrous tip Temporosphenoidal or cerebellar otitic abscess may give rise to deep, constant boring headache, caused by increased intradural pressure

Pain may, of course, reach the ear by any or all of the routes named simultaneously It will be noted that these pathways intermingle closely with those involving the nose, pharynx, accessory sinuses, and larynx

Aural Neuroses and Psychoses.—Establishment of "pathways of pain" often repeated or continuous from the nose, jaw, or throat to the ear may give rise to associations with and rationalizations from extraneous circumstances, such as fear of invasion by cancer, or of permanent and total loss of hearing Tinnitus, associated with eustachian tubal blockade or with adhesive middle ear processes, as well as tinnitus in otosclerosis, may lay the foundations

for worry, sleeplessness, and the eventual building up of faulty interpretation of the sounds into "overheard voices," "ringing bells," hostile whispering, threats of impending doom, and other delusional concepts. Such difficulties are more frequently found in association with arteriosclerotics, women at the climacteric, or in poorly oriented adolescents. While phenomena arising from vestibular congestion or bemorhage may include severe vertigo and great prostration, neurotic or psychic upsets from this cause are rare. Such people are too sick to build up imaginary explanations for their symptoms, although with repeated attacks they may manifest extreme terror and discomfort.

Confusional states, interference with attention, mutism, and stuttering are not only found associated with severe or total deafness in childhood, but are incidental as well to bombardments and other explosive traumatism to the auditory apparatus.

Nasal Neuralgias, Headaches, and Neuroses
—Neuralgias and Headaches from Mucocutaneous Irritations—Nasal neuralgias include (1) those arising from sensory nerves which pass through the sphenopalatine ganglion (itself sympathetic in character, however), (2) those originating from the nasociliary and anterior and posterior ethmoidal branches of the ophthalmic division of the trigeminus, (3) those coming from the palatine and superior dental branches of the maxillary division of the trigeminus, (4) pain due to localized ischemia, caused by sympathetic irritation in the sphenopalatine region.

Narrowed nasal passages, whether by turbinate swelling (inflammatory or allergic), thickening or displacement of the nasal septum, or by neoplasms, are responsible not only for direct nerve irritations felt along the above routes, but also for closure of sinus ostia, with retention of normal mucus or of eventual inflammatory exudates. Headaches arising from such retention are referred to the cutaneous trigeminal distribution as follows: Frontal sinuses—above the eyes, to the vertex and temples, anterior ethmoids—pressure between the eyes, and to the temples, posterior ethmoids and sphenoids—in front of and behind the ears, occasionally to the occiput and shoulder, maxillary antrum—above the eye, and to teeth of the same side.

The nasociliary nerve lies in close relation to the nasofrontal duct, hence, closure of this duct

by neoplasms (osteoma, mucocoele) may provoke severe pain running down the nasal bridge. Trauma to the nasal bones at their articulation with the frontal often injures the nasociliary nerve and includes it in an eventually painful scar.

The anterior and posterior ethmoidal nerves, and fibers from sphenopalatine connections with the trigeminus, are closely related with the orbital contents, and thus ethmoidal congestion may lead to retrobulbar pain, lacrimation, and blepharospasm.

Stimulation of the sphenopalatine region, as by inflammatory exudates, crusts, or swellings, has been found to cause painful ischemia of the skin overlying the mastoid, suboccipital, and trapezius regions. Such nasal irritation is picked up through the great deep and superficial petrosal nerves, giving rise to stimulation of reticulospinal cells in the medulla, which activate the sympathetic vasoconstrictors in the lower cervical and upper thoracic regions. These painfully diminish the arterial supply of the ear and side of the neck, and may, through local anoxemia, excite partial spasm of the trapezius muscle.

Disturbances of Olfaction—Olfactory receptors in the limited mucosal area of the upper part of the nasal septum and the corresponding section of the superior turbinate may readily be shut away from contact with odoriferous particles by swelling of the middle turbinate. If this swelling be inflammatory and long continued, it may extend upward into the olfactory mucosa and thus, eventually, through metaplasia and fibrosis, may lead to destruction of the olfactory cells. Anosmia is thus not infrequent in cases of nasal allergy, chronic ethmoiditis, and ozena.

During the progress of such inflammatory changes, perversions or alterations of the sense of smell (parosmia) may occur, and hypersensitivity to certain odors may develop. Cure of the underlying nasal disorder, often associated with endocrine imbalance, will reopen the upper nasal meatus and olfactory groove to normal olfaction unless permanent tissue changes have taken place. Kakosmia (apparent perception of nonexistent evil odors) may occur during metaplastic changes in the olfactory mucosa. All these olfactory changes may, because of the close association of smell and taste, give rise to the notion that food has "lost its flavor," or that it "tastes bad." Kakosmia may also be a

transitory symptom (or aura) suggesting the possibility of circulatory or neoplastic changes in the uncinate gyrus of the brain

Complaint of persistent bad odors (and tastes) may lead to hallucinations and paranoid delusions. The subject may believe that an attempt is being made to poison him with gases or food

Other Nasal Neuroses—Spasmodic attacks of sneezing are somewhat comparable to hic cough, facial tics, and other habit spasms. They may be induced by cold air, by pungent inhalations, or by touching the tuberculum septi with a wisp of cotton. Not infrequently, they are associated with nervous stresses—embarrassment, sudden emotion, worry. They are usually found in emotionally unstable persons, sensitive, timid, overwrought, reacting poorly to environmental change. Such individuals are found among adolescents, women at the true or operative climacteric, overworked business men, "stale" aviators, and military inductees. There is no demonstrable organic basis for this annoying symptom.

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EXAMINATION OF THE EAR AND OF THE FUNCTION OF HEARING

Patients present themselves for examination of the ear or ears either for an acute condition, often associated with pain, or as a result of some chronic condition which has gradually reached the point where some type of inconvenience develops, either discomfort or a hearing loss. Minor difficulties, unfortunately, are usually disregarded. When the patient finally seeks examination and treatment the otologist may be sure that his services are required.

A careful history is of course the essential preliminary approach. Familial tendencies, childhood diseases, upper respiratory infections, surgery, general health, habits such as smoking, drinking, drugs, dietary excesses or privations must all be recorded. In addition climatological influences, occupational factors, age, sex, and race, all offer important presumptive material for the final diagnostic judgment.

A thorough analysis of the social, economic, and psychologic adjustment of the patient, where hearing impairment is the predominant factor, must always be the preliminary step before physical examination begins. More obvious estimations are the appraisal of the patient's voice and speech characteristics, and the intensity of his speech—loud if the hearing loss is perceptive, soft if the conductive type. In long-standing impairments, even in adults, speech takes on certain abnormal characteristics.

Furthermore, the otologist must know the patient's general constitutional status with particular reference to the cardiovascular and endocrine systems and to allergic manifestations. Blood examination and neurologic study are essential parts of this initial survey. The routine Wassermann in chronic ear conditions of whatever kind will seldom be positive but its neglect will occasionally lead to serious embarrassment, both for the patient and the physician.

With the data obtained from this type of examination the otologist may then approach his otoscopic examination with a truly seeing eye.

Otoscopic Examination—The physical examination of the ear may be divided into two parts which will be called external and internal otoscopy. The former seldom reveals data of significance and where abnormalities are present the history will have supplied clarifying details. The external ear or auricle is susceptible to hereditary peculiarities of contour, a matter of little significance so far as hearing is concerned. Every degree of congenital defect may be present from complete absence of the auricle and atresia of the external canal to any less abnormal type of development.

Acute trauma to the auricle should be easily recognized as should any type of acute infection. The reaction to this latter, due to the peculiar anatomy of the auricle, is unmistakable. When infection is present the post-auricular glands are often involved. In all acute infections of the auricle or external canal, pain is a subjective factor and is elicited on the

slightest pressure either by traction on the pinna or by the introduction of the speculum into the canal.

The otologist is concerned primarily with internal otoscopy. By this is meant the view obtained of the external auditory canal and tympanic membrane by means of brilliant reflected light through an otoscopic speculum fitted with lenses which will provide magnification of at least one diameter and preferably three. Without magnification of some degree internal otoscopy is of little value aside from the recognition of an acute middle ear inflammatory process and many early and significant anatomic changes will be missed. Whenever possible some type of "optical loop" should be employed. There is no substitute for binocular vision either in examination or treatment of the ear.

The External Canal—In a dark room with adequate illumination of the ear the speculum is introduced into the external meatus of the canal. Ordinarily an immediate view of the tympanic membrane is available. If this is not the case obstruction to such an inspection is usually caused by cerumen, abnormality in contour and direction of the canal, exostosis, an acute inflammatory condition, a new growth, and/or a foreign body.

CERUMEN—Small accumulations to complete impactions are frequently found.

ABNORMALITIES IN CONTOUR AND DIRECTION OF CANAL—This is a relatively frequent occurrence.

EXOSTOSES—These bony outgrowths range in size to produce partial or complete obstruction of the canal, are relatively frequent, may be single or multiple, occur almost invariably in man and may constitute a serious hazard. They have been ascribed to the effect of cold water on the ear but from the author's experience are related more specifically to the so-called "gouty diathesis" or lacking this fast disappearing malady the obstructions seem to appear more often in the ears of individuals who have maintained for years a high alcoholic intake.

ACUTE INFLAMMATORY CONDITIONS—This is probably the most frequent obstructive lesion to be seen. Single or multiple furuncles, cellulitis of the canal, eczema of the canal wall (allergic), and complete occlusion of the lumen by purulent discharge from an acute or chronic suppurative otitis media may all interfere with

adequate inspection. The typical reaction to a mycotic infection is an important and relatively easily recognizable condition. Profuse discharge, large plaques of desquamated epithelium, and resistance to routine treatment are diagnostic in addition to the final cultural identification.

NEW GROWTH—Benign polyps of exuberant granulation tissue are of fairly frequent occurrence. They may vary in size from a small protrusion through a chronic perforation to a mass which may fill the entire canal and sometimes even protrudes through the external meatus. They are almost invariably bathed in pus. Malignant new growths of the external canal are unusual, probably less than one in a thousand cases and are recognized by the same character and appearance which is found in malignant growth or ulceration elsewhere in the body.

FOREIGN BODIES—The possibility of foreign bodies in the external canal should always be borne in mind, particularly, of course, in children. A list of such objects would include insects dead or alive (from June bugs to gnats), pencil and crayon points (these are frequently found), beads, glass, peas, beans, and corn—in short any object of a size permitting its entrance into the external canal. Such foreign bodies are often difficult to recognize and may be even more difficult to remove. Although treatment is not a part of this particular discussion, removal of any obstruction is obviously essential for adequate examination of the tympanic membrane. At this point attention must be directed to the fact that the lining epithelium of the external canal is extremely delicate and susceptible to trauma. In all attempts to relieve obstruction of whatever kind the most delicate instrumentation should be employed. To remove foreign bodies special instruments are necessary such as angled alligator forceps or punches. If these instruments are not available the physician should not attempt removal but refer the case to a specialist. In the presence of an obstruction damage to the canal wall may prove disastrous. It is far better to wait until all conservative measures have been employed before attempting to remove an obstruction by any type of radical procedure. This statement applies even to the removal of an impacted mass of cerumen.

The Tympanic Membrane—The reader is referred to the section entitled "Anatomy of the

Ear' for a description of the normal appearance of the tympanic membrane. It may be assumed, however, that few patients requiring otologic examination will have normal tympanic membranes.

Abnormalities related to pathologic conditions include thickening, retraction, reduction in size, or absence of the light cone, immobility of the membrane, scarring central or marginal sclerosis, acute or chronic perforation, and absence of tympanic membrane.

1 **THICKENING**—This condition may be present in every degree of severity from change of a normally delicate tympanic membrane precluding easy visualization of middle ear structures to the extreme in which the membrane has an almost cartilaginous appearance. The entire membrane may be involved or simply localized areas.

2 **RETRACTION**—This change may be either acute or chronic, more commonly the latter. The degree of retraction may vary from a slight concavity of the membrane to an almost funnel-like contour. In extreme situations the membrane may actually be adherent to the promontory of the cochlea.

3 **REDUCTION IN SIZE OR ABSENCE OF THE LIGHT CONE**—Size and brilliance of the light cone depend upon the brilliance of the illumination. With any standard lighting the otologist will learn to appraise changes in this reflex from reduction in normal brilliancy to complete absence. Such changes are dependent upon the degree of thickening and retraction of the tympanic membrane in most instances.

4 **IMMOBILITY OF THE MEMBRANE**—This is determined by any method desired (examination with Siegle's otoscope, for example).

5 **SCARRING**—This gives evidence of a previous infectious process.

6 **CENTRAL OR MARGINAL SCLEROSIS**—The latter condition is usually present in older age groups and is comparable to the arcus senilis of the eye.

7 **ACUTE OR CHRONIC PERFORATION**—Here history is an essential factor. Perforations may occur in any portion of the tympanic membrane. Central and posterior perforations are the most common—the posterior because this is the site usually elected for myringotomy, the anterior is usually the result of spontaneous or multiple perforation. In the latter case systemic disease should be considered.

8 **ABSENCE OF TYMPANIC MEMBRANE**—This

situation is due to long-standing chronic suppurations or radical mastoid surgery. The tympanic membrane is absent except for some small remnant around the annulus, and the middle ear cavity is covered with thickened mucous membrane or granulation tissue. Few landmarks are recognizable. There may be a slight indentation in the surface contour over the position of the round window or a slight prominence over the position of the oval window if a sequester of the stapes is still present. The middle ear cavity may be wet or dry. If wet, the hearing loss is relatively moderate, if dry, the loss is the extreme of a middle ear impairment, 50 to 60 decibels.

Special Tests—No direct examination of the ear can be complete without roentgen ray examination of the mastoids in all cases of infection whether acute or chronic. When roentgen-ray examinations are made the petrous ridges in addition to the mastoid bones should be carefully scrutinized.

Complete blood counts should always be made. This is particularly important as a matter of record when sulfa therapy is to be instituted. Routine Wassermanns are highly important.

Although vestibular tests have been discussed elsewhere their importance may be emphasized here. Vastine¹ has postulated, "As goes the labyrinth, so goes the cochlea." The caloric test demonstrates easily and immediately an intact static labyrinth. Demonstration of a viable or nonviable labyrinth is the matter of chief concern.

Having completed every direct and indirect type of examination of the ear its functional ability may now be properly explored.

Examination of the Function of Hearing—Since the discovery of the thermionic tube and its eventual association with electrical circuits designed to produce measured frequency cycles, audiometry, or the method of measuring the acuity of human hearing, has become an exact science. Previously, sound sources for this important determination consisted primarily of tuning forks and the whispered and spoken voice, calibrated with some accuracy in terms of duration but quite inaccurately in terms of measured loudness. The development of audiometry, which Bunch² described so completely, represents primarily a reaction to the obvious inadequacies of the earlier methods of testing hearing. The introduction of the vacuum tube audiometer, with its specific frequency and vol

ume ranges, marked a new era in scientific methods for the appraisal of hearing loss.

Audiometry today is an accepted otologic technic. It measures specifically and accurately impairment of hearing against an established normal level for both air and bone conduction. Its scope is rapidly expanding. No longer is the simple establishment of "degree" of deafness adequate. Special technics have contributed qualitative and quantitative information essential for diagnosis of any type of hearing loss. However, the discrete frequency audiogram, sometimes improperly called "pure tone," is now generally accepted as the measure of hearing acuity. It measures the improvement resulting from any type of therapy. It is the basis for the much discussed percentage loss of hearing.

There is no need to repeat in this description of functional examination of the ear the many routine tests carried out with tuning forks, watch tick, Galton whistle, monochord, and whispered and spoken voice. They have all been described in detail innumerable times and have no place in any modern treatise on audiometry. Great names in the specialty of otology have figured in the development and routine procedures of appraising hearing losses. The time has now come to abandon surnames for tests applied and have examinations named according to their diagnostic purposes.

Audiometry may be defined as the measurement of the ability of the human ear to perceive individual tones or to understand speech at minimal intensity levels. This level is called the "threshold of hearing" and is the faintest intensity of discrete frequencies or speech which the ear under examination can hear with accuracy.

Audiometer—An audiometer is an electrical instrument which is designed to deliver a series of discrete frequencies in measured intensity steps to the ear under examination. These stimulating tones consist of five or six octaves above middle C—256 cycles—and one below it. The frequency 64 cycles has now been abandoned. The component parts of the audiometer are as follows:

1 OSCILLATOR—Based on the performance of vacuum tubes and related electrical circuits the oscillator produces tones of any desired frequency within the audiometric range.

2 FREQUENCY SELECTOR—In discrete frequency audiometers the frequency selector automatically changes the tone delivered in

octave or half octave steps. Sweep frequency instruments deliver the complete band from the lowest to the highest tone available.

3 ATTENUATOR—This is the unit of the audiometer which controls the intensity of the tone delivered to the ear in steps of 5 decibels. The attenuator must perform accurately at all levels of the instrument's intensity range.

4 RECEIVER—The receiver converts the electrical impulse into an acoustic stimulus which is applied to the ear under test.

At this point the availability of audiometers might be discussed. Obviously the general practitioner has no real use for this particular piece of apparatus. By the same token he is not qualified to pass judgment on the extent of any hearing loss, particularly in children. Whenever any case presents itself with even a questioned loss of hearing the patient should be referred to an otologist who has available adequate means of examination. Grave injustice has been done to individuals with a hearing loss by the general practitioner's easy assumption of authority in this very complex problem. School children are misunderstood and assigned to the "dull group." Teachers fail to relate speech defects to a hearing impairment. In individuals of all ages, pediatricians, medical consultants, and psychiatrists are very largely oblivious to the implications of a handicapping hearing loss. Psychologic reaction, nervous instability, and profound depression are all related manifestations. The otologist at the present time is the only one who appreciates adequately the remote effects of a handicapping hearing loss.

Types of Hearing Loss—Essentially there are three types of hearing loss: (1) the uniform loss with more or less equal impairment for all frequencies, (2) the high tone loss with relatively normal hearing up to 1024 cycles with a progressive impairment towards the highest measured frequency, (3) the predominant low tone loss, the least common of the three, in which low frequencies are impaired to a greater extent than the high.

This broad grouping quite naturally serves only as a basic pattern. Departures from it may be in any point of the frequency range, in unexplainable dips and peaks, in bizarre variation in pattern, and above all in individual reaction to what might otherwise be considered a typical hearing loss audiogram.

Many authors and a few investigators have developed classifications of hearing loss. These

tabulations are all essentially the same with points of departure, one from the other, based more on some personal interpretation than any fundamental factor of hearing. The personality of the hard of hearing or deaf person is rather consistently ignored in the general effort towards specific classification. So long as this approach to the problem of hearing loss persists little of real consequence will be accomplished. The hard of hearing or deaf person will always be individual in his reactions. He can never be treated simply as one of a group

moderate the audiometer, a table, and two chairs without undue crowding. The subject's chair should be an arm chair and reasonably comfortable. A room 6 by 8 feet is entirely adequate. The door should be tight fitting and preferably weather stripped.

OPERATION OF AUDIOMETERS—Air Conduction—The subject is seated so as not to be able to see the operation of the audiometer, with his back to the audiometer and examiner. He is instructed to press the indicator button whenever and as long as the signal is heard.

AUDIOGRAMS

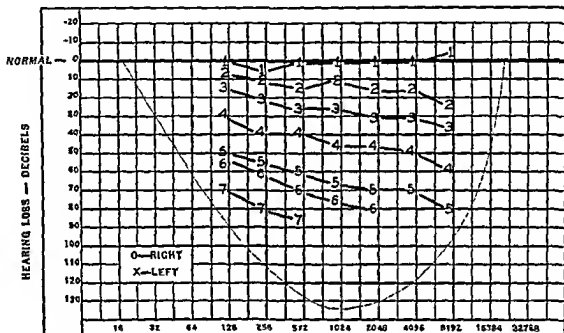


Fig 203—This chart illustrates the type in general use. If all the symbols 'o' and 'x' fall within ten times above or below the zero or normal line the individual is considered to have normal hearing. But as the hearing becomes more and more impaired the symbols will fall further and further below this normal level. On this chart are represented a series of typical audiograms labeled as follows: 1, normal hearing; 2, slight impairment; 3, recognized impairment, but no real handicap; 4, beginning handicap; 5, serious handicap; 6, complete inability to hear or understand the spoken voice; 7, no usable residual hearing.

Technic of Audiometry—CONDITIONS FOR EXAMINATION—All examinations should be carried out in soundproof rooms. These are not generally available and as a substitute a quiet, reasonably well ventilated room remote from street, corridor and plumbing noises should be chosen. Curtain hangings will add to the sound conditioning. The average noise level in a quiet office is from 30 to 40 decibels. This should be reduced to 20 decibels or less. Roughly such a level is achieved when to the normal ear no extraneous sounds, such as typewriters, conversation levels, and annunciators, are audible. The size of the room should be sufficient to accom-

The button is released when the signal is interrupted.

It is now accepted practice to start the audiogram with the frequency 1024 cycles proceeding in sequence to the highest frequency available on the instrument. This standard is not simply an arbitrary one but is based on the fact³ that response at 1024 cycles is the most accurate in the entire audiometric scale and it is the least subject to variation. From a diagnostic standpoint, the high frequency response is of significant importance, the three lower frequencies are relatively unimportant. Fatigue occurs more at the examination of the high frequencies than at

the low, therefore the high frequencies should be examined first. The operator then returns to 512 cycles and in order examines the responses for 256 and 128 cycles.

A reasonably loud signal should be presented first, basing the intensity on the apparent hearing loss. Attenuation is introduced in 5 or 10 decibel steps until no response is indicated. Attenuation is then taken out until the tone is heard. This maneuver should be repeated and

frequencies it should be held firmly and accurately against the ear. Before recording a high tone (ooo above 4096) as not being heard, the threshold level should be checked in relation to the standing wave effect—that is, the receiver should be moved away from and towards the ear over a distance of $\frac{1}{2}$ to $\frac{3}{4}$ of an inch, if the specific frequency is not heard and if heard at any significant departure from normal this check should always be employed.

[illegible]

Fig. 204A—Form for recording air conduction audiograms. When repeated audiograms are to be made for the purpose of gauging the results of therapy, this type of recording form permits immediate evaluation of changes in the hearing level. It eliminates the frequent inaccuracies which occur in rapid recording on chart forms, and also saves space.

a uniform response obtained at least three times for each frequency. This level is recorded as the threshold of hearing for the frequency involved. The interrupter button should be used whenever the attenuator dial is changed and the length of time the tone signal is presented and the interval between signals should be constantly changed thus avoiding any possibility of rhythmic response. This is particularly important when examining children.

Care should be taken to check the fit and position of the audiometer receiver. For the low

Bone Conduction—It must be understood at the outset that all methods of determining the bone conduction acuity of the ear are purely relative. This statement holds both for examination by tuning forks and electrically activated bone conduction receivers.

A satisfactory normal threshold of hearing of the human ear by bone conduction has never been established. However, the electrical receiver has many advantages, chief of which is its readily available frequency range—*that of the audiometer with which it is used*. Normal

bone conduction is known to be 30 to 40 decibels less efficient than normal air conduction. Audiometer scales of threshold levels are calibrated on this basis. With this factor in mind it is a simple matter to determine whether the Rinne test is positive or negative. The Weber lateralization test is carried out with equal ease.

Bone conduction acuity is essentially a differential diagnostic factor. It is not an absolute

diagnosis of unilateral hearing impairment and the only means whereby a complete monaural deafness can be established. It is an established fact that attenuation from the ear under examination to the opposite one, provided both are normal, is approximately 30 decibels by air conduction. Therefore, if one ear has normal acuity and the other is impaired 30 decibels or more the subject may be responding via the better ear.

[illegible]

Fig. 204B — Form for recording bone conduction audiograms, lateralization, and loudness balance data

measurement Any otologist possessing an audiometer can readily establish a base line normal for his particular instrument for bone conduction Departures from this line will supply the diagnostic factor sought

Masking—One of the most important and at the same time least used and understood than any other audiometric procedure is auditory masking. It is an essential factor in the actual

and the response from the poorer is necessarily not a true measure of the ear's actual acuity. Whenever audiometric levels for the two ears depart from each other by 30 decibels or more and conform in their general outline the result is spoken of as a "shadow curve." When this situation is present, "masking" is an essential procedure.

By masking is meant the reduction of the

normal acuity of an ear in the presence of noise. When deliberately applied the masking noise should be controllable as to intensity in 5 decibel steps and should be of such character that it embodies in its frequency range the entire audiometric scale. The majority of masking devices (Barany, Larmapparat, electric buz

An air jet can serve the same purpose but is of course, uncontrollable as to actual loudness. When careful audiometric examinations are being carried out every effort should be made to secure a proper auditory masker.

Only a few of the modern audiometers have incorporated in them such a masking device and

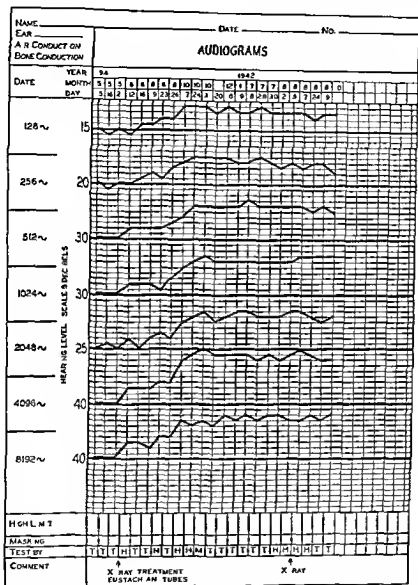


Fig. 205—Continuous audiogram chart. The data which would be recorded on form shown in Figure 204A can graphically demonstrate the change in hearing level resulting from therapy. The base line level for each frequency is determined by the first audiogram made. The vertical blocks represent 5 decibel steps.

zers, etc.) do not fulfill this stipulation as they have essentially a low frequency response and cannot mask high frequencies except at undue intensity.

The thermal hiss of a vacuum tube suitably amplified and calibrated is the ideal masking noise since it consists of a wide frequency band

this is essentially only a buzzer system and consequently of no real value. Individual models are available which give adequate frequency response and every effort should be made to secure such a unit. If this also is impossible an air jet gives the most satisfactory masking noise even though it cannot be calibrated as to in

tensity. However, when understanding of speech with a normally hearing ear becomes confused in the presence of noise that noise level may be assumed to be approximately 60 or more decibels above threshold which would be a reasonably intense masking level.

For air conduction the intensity of the masking noise should be 30 decibels plus the actual disparity in the threshold levels of the two ears. Under most circumstances a noise level of 70 decibels above normal threshold is adequate. Eighty-five decibels may sometimes be neces-

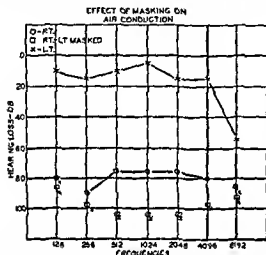


Fig. 206—Without masking, this patient apparently had a hearing level in the deafened ear of approximately 80 decibels below normal. With appropriate masking a complete loss of hearing was demonstrated. In this case the entire static and acoustic labyrinth had been destroyed by a sarcoma of the submaxillary fossa.

sary to demonstrate a complete unilateral loss but intensities greater than this may mask the poorer ear by bone conduction and should not be used.

In bone conduction examinations the ear not under test should always be masked regardless of any disparity between the two ears. To determine the intensity of masking, the following factors should be considered. Attenuation through the head by bone conduction is 15 decibels. To this is added the 30 decibels air conduction factor mentioned previously plus the air conduction decibel difference in the acuity of the two ears. The sum of these figures should be the loudness of the masking tone, but an intensity greater than 75 decibels should never be employed for bone conduction.

Special Tests—LOUDNESS BALANCE TEST—One of the most important special diagnostic procedures is the "loudness balance test." The

theory and experimental basis for this test has been thoroughly described elsewhere (Steinberg and Gardner,⁴ Fowler,⁵ Lorente de No.⁶ The general principle of the test is as follows. When

LOUDNESS BALANCE

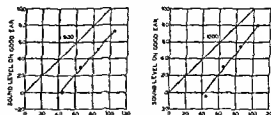


Fig. 207—Chart of a constant loudness balance test. The straight line represents level of normal or reference ear. The charted line showing initial disparity of 40 decibels maintains this difference up to an intensity level 80 decibels above the threshold for the two frequencies used. This demonstrates a conductive type of deafness.

there exists a disparity between the hearing level of the patient's two ears it must be determined whether the poorer hearing ear pre-

LOUDNESS BALANCE

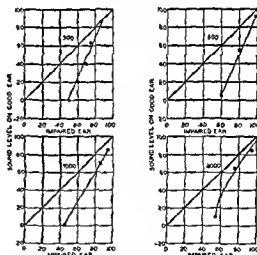


Fig. 208—Chart of a variable loudness balance test. This is similar to Figure 207 with the exception that at an intensity level of 85 decibels above the threshold the charted line intersects the reference line. In other words, the impaired ear is equal in acuity to the better ear at this level. This demonstrates a perceptive type of deafness in the impaired ear. (Republished by permission from the Annals of Otology, Rhinology and Laryngology 47: 78, 1938.)

sents a conductive or perceptive type of deafness. This test is the only absolute method in the differentiation between conductive and perceptive deafness. If it demonstrates that the

disparity remains constant with any degree of loudness, the loss is a conductive one. If perceptiveness, the poorer ear hears with equal acuity tones at maximum loudness. Where bone conduction findings are equivocal, the loudness balance test will invariably resolve the difficulty. There is no need to detail the technique of the procedure here other than to say it is entirely practical, is not a burdensome test for any patient, and not unduly time-consuming for the examiner. It should and undoubtedly will receive much more general application when its advantages and significance are more generally appreciated and the otologist is willing to procure the additional equipment necessary.

FATIGUE TEST—Clinical fatigue of the ear is another differential factor between conductive and perceptive deafness. This is a direct application of experimental findings. Under all reasonable intensities of sound stimulation an ear impaired by involvement of the conductive mechanism will not fatigue, whereas, in a case of perceptive deafness, fatigue will develop if the ear is subjected to a frequency of high intensity for periods of time ranging from five to ten minutes. Sound sources capable of producing intensities 80 to 90 decibels above threshold must be available for the proper performance of the fatigue test. With the audiogram as a base line the ear is exposed to a fatiguing tone of the proper intensity using preferably the frequencies 512, 1024, or 2048. The audiogram is repeated immediately after the ear has been fatigued. If a lowering of threshold of 10 decibels or more is demonstrated actual fatigue has occurred. It should be noted that fatigue occurs at the level of the fatiguing tone and above it but never below.

SPEECH RECEPTION TEST—"However standard the use of the discrete frequency audiometer has become as a method of measuring hearing loss, it still leaves many questions unsettled. Chief of these is the relation of this threshold audiogram to the practical disability imposed by a given hearing loss. 'We do not live in a world of threshold sound' is the commonest criticism advanced, 'The relationship between the audiogram and ability to understand speech is obscure' is another. The ability to hear represents the most vital of all human contacts. Without this sense, verbal communication, the basis of all social intercourse, is eliminated."⁷

Three methods for determining this ability to understand speech are available and may be briefly described as follows:

1 *The spoken voice is controlled electrically with an accurate attenuating system, and presented indirectly to the listener by means of a high fidelity loud speaker.* This method requires equipment which will be found in only a few medical or acoustic laboratories where a sound-proof room, in which speech can be presented to the subject by means of a loud speaker, is available. The intensity of voice must have a calibrated attenuating control capable of modifying loudness in steps of 5 decibels or less over a range of 80 decibels above normal threshold. Beyond this point most electrical equipment tends to overload and distortion is introduced. A list of sentences is read by a speaker through a microphone and these are transmitted through an amplifying system. The intensity of the speaker's voice is modulated by observing the excursion of a decibel meter indicator needle. (The sentence lists used are those devised by the Bell Telephone Laboratories and comprise 50 lists of 50 sentences each, and are to be found in *Articulation Testing Methods*, H. Fletcher and J. C. Steinberg, Bell Telephone Laboratories, Inc., Reprint B-436, November, 1929.) As in standard audiometry the subject records accurate understanding of speech by establishing a level of accuracy of at least 60 per cent. Here accuracy is determined by repeating the presented sentences verbatim instead of pressing the signal for heard or not heard as in the audiometric examination. Sufficient time is allowed between the presentation of each sentence for the subject to repeat what he has heard without undue hurry. Each sentence has a key word or words, which are critical in the correct repetition of the sentence. Interestingly enough this method is accurate to plus or minus one decibel and does not introduce any element of deterioration in production and is completely flexible, both of which factors must be taken into account in the following method.

2 *The spoken voice is presented through an electrically controlled circuit but phonograph records are used as the sound source.* The general electrical system and physical surroundings are essentially the same as those described for the first method. One less in the examining personnel is required. This method, however, is entirely inflexible and subject to record deterioration due to repeated playing.

3 The spoken voice is presented through a roughly calibrated nonelectrical attenuating sys-

NAME E M B

SPEECH RECEPTION TEST

DATE	3 24 41	
RECORDER	ET	
HEADER	RK	
LIST NO	4	
NOTE	Normal Ear	

ATTENUATOR SETT NO	SENTENCES	
	CORRECT	NCORRECT
30	111	
40	111	
50	111	
60	111	
70		1111
65	1 111	
66	111	11
50	111	
67	11	111 11
66	111 111	1

[illegible][illegible][illegible][illegible][illegible]

Fig 209—Charting form for speech reception test. The figures under attenuator setting are the intensity levels of speech delivered to the ear under examination. It will be seen that as attenuation is increased in decibel steps a certain number of sentences were not repeated correctly. At the 66 decibel level eight sentences were repeated correctly and one incorrectly. This value is taken as the threshold of speech reception.

tem the general intensity levels of which can be reasonably well controlled and estimated. If the forementioned equipment (methods 1 and 2) is

pering The natural reaction of every speaker is to raise his voice as he departs from the listener Any standard 2 foot megaphone increases the intensity of the normal voice at the listener's ear by approximately 20 decibels If normal speech is of the order of loudness of 40 decibels above threshold, and to this added an additional 20 decibels, few impaired hearing levels would fail to be reached through such a system Attenuation in the system can be introduced by a series of felt disks graded in diameter to that of the megaphone at the point of their placement within the megaphone Such a system can readily be constructed so that each felt disk will introduce an approximate attenuation of 12 decibels If four disks are placed the total loudness range of the device is 50 decibels A person with normal hearing can understand speech with the megaphone at a distance of 6 feet from the ear with all the felt disks in place The speech reception level of the deafened individual is determined by removing the disks and, if necessary, shortening the distance between the megaphone and patient The mouth-piece of the megaphone must be circled with some type of sponge rubber which will prevent escape of sound outside of the megaphone

Summary.—Although the purpose of this review is to outline very briefly the physical and functional examination of the ear, attention must be drawn to the importance of the procedure It is not simply an appraisal of a hearing impairment It is a highly important diagnostic method Cerebellopontine angle tumors, tumors of the auditory nerve, and vestibular derangements, such as Meniere's disease and its many atypical manifestations, depend entirely for complete diagnosis upon careful functional examination of the patient's hearing level and the reason for the impairment

Emphasis must be directed to the fact that audiometry is a diagnostic procedure It is the indication for treatment or the use of a hearing aid It is the only means by which the effectiveness of therapy can be gauged and unless careful audiometric examinations are carried out and improvement in hearing is a matter of actual record the results of any therapy are entirely inconclusive The patient's subjective reaction cannot be accepted as a true measure of the effectiveness of treatment Audiometry as here described is a precise, direct, and accurate procedure It eliminates the subjective euphoria so deluding to the patient and physician Finally,

it places a specific curb upon the modern and indiscriminate use of unestablished forms of treatment

WALTER HUGHSON

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DISEASES OF THE EXTERNAL EAR

The external ear, which consists of the pinna and structures of the auditory canal, may be the seat of a multiplicity of pathologic conditions—traumatic, inflammatory, infectious, and degenerative Their characteristics are the same as those of similar lesions occurring elsewhere in the body, but there are morphologic variations due to the slightly different texture of the skin and the presence of the ceruminous glands which secrete a sticky substance, the cerumen A good working knowledge of dermatologic lesions as they occur elsewhere in the body will materially assist in understanding and treating lesions of the external ear The presence of cartilage underneath the skin in this region plays an important part in the clinical course of many lesions The exposed position of the external ear makes it vulnerable to temperature changes, traumatism, insect bites, and the effect of the elements The external auditory canal varies greatly in size and shape, and the size of hairs in the skin of this region, as well as their number, is also subject to considerable variation It is a skin-lined cul de sac, which acts admirably as a culture tube for the growth of

pyogenic and nonpyogenic organisms as well as certain pathogenic molds. In the depths of the canal is the ear drum, the outer surface of which is covered with stratified squamous epithelium identical with that of the remainder of the canal, but differing greatly in texture and thickness. The skin of the canal, as well as that of the external ear, or pinna, is closely attached to the underlying cartilage and bone, which explains the extremely painful character of certain lesions such as furuncles, as there is little room for expansion to take place.

Traumatism.—Wounds affecting the external ear are classified as contused, incised, punctured, and lacerated, the same as elsewhere in the body. They are either infected or non-infected, this depending largely on the manner in which they were incurred. Loss of substance may occur, ranging from slight defects to complete avulsion of the part.

The general principles governing the treatment of wounds elsewhere in the body apply to the treatment of wounds of the external ear. Hemostasis, antiseptic, and proper closure are to be observed. Debridement, if indicated, should be performed with conservation of every bit of tissue possible. Defects may be corrected by subsequent plastic procedure. The recent adoption of a sulfonamide powder as a local first-aid measure as well as a wound dressing has materially lessened the incidence of wound infections in both civilian and military practice.

Hematoma of the Auricle (Othematoma, Prize Fighter's Ear, Cauliflower Ear).—This condition, in which there is extravasation of blood beneath the skin or perichondrium, of the external ear, results from trauma. The accumulation of blood, if not removed promptly, becomes converted into fibrous tissue, and subsequently into cartilage, giving rise to a permanent and unsightly deformity. Blows inflicted with the fist are the most frequent cause, therefore, hematoma of the auricle is of common occurrence in prize fighters, the so called cauliflower ear being regarded more or less as the badge of the prize-fighting profession. Spontaneous subperichondrial hemorrhages are said to occur in inmates of institutions caring for patients with mental disorders, but it is believed that careful investigation of surrounding circumstances will in most instances reveal the fact that there is usually some trauma underlying the condition, even though it may be the result of only a gentle slap from the hand of an

unsympathetic attendant or another inmate. I have never encountered an undoubted instance of spontaneous subperichondrial hemorrhage of this nature, and diligent inquiry has failed to uncover a single authentic instance occurring in inmates of institutions caring for persons with mental disorders.

The use of a suitable shield over the ears in prize fighters, wrestlers, football players, and others likely to receive blows on the external ear will usually prevent the occurrence of a hematoma in this location.

Hematoma of the auricle must be differentiated from serous effusions underneath the perichondrium such as occur in association with frost bite, or in the course of an acute perichondritis. Aspiration and/or transillumination will reveal the nature of the fluid accumulation.

Treatment consists in removal of the accumulation of blood. All surgical maneuvers must be conducted under standard conditions of antiseptic and aseptic. Prompt care obtains best results. If treatment is sought sufficiently early while the extravasated blood is still fluid, aspiration with a moderately large, short needle may be tried, but such an attempt is usually fraught with disappointment. Better results are obtained by puncturing into the swelling with a cataract knife in one or more places, and expressing the blood manually or introducing a blunt-pointed cannula through the punctures and aspirating the accumulated fluid. The most satisfactory procedure in my experience consists in making one or more incisions parallel to the border of the helix, usually a short one anteriorly and a longer one posteriorly down to and through the perichondrium if necessary, and evacuating all blood and clots (the blood is usually firmly clotted by the time an otologist is consulted) by applying pressure and using a curet (the ordinary large sized chalazion curet [Myerhofer] is very satisfactory, or the non-cutting ring type ear curet may be used). Quite often the thickening about the ear is not due so much to extravasation of blood underneath the perichondrium or skin as to actual infiltration of the skin with blood. In the latter instance the contour of the ear can only be restored by raising up the flap of skin by making a more liberal incision and by excising the deeper portion into which infiltration has taken place. Apposition of the skin and perichondrium is obtained by gentle pressure and the use of a mold of dental

modeling compound plaster of paris or paraffin. Gentle pressure must be maintained until the danger of recurrence of bleeding has passed and adherence of skin and perichondrium to the underlying cartilage is assured. Too great pressure may lead to necrosis of the skin and must be guarded against. The use of parathyroid extract hypodermically 0.5 cc. once daily, and calcium gluconate or levulinate 15 grains or 1 gm. intravenously is of service in controlling the recurrence of hemorrhage. Vitamin K may also be used orally to good advantage. The three preparations may be used jointly.

Thermal Effects (Dermatitis Calorica).—Under this heading are classified the effects produced by extremes in temperature either heat or cold.

Frostbite or Freezing (Dermatitis Congelationis).—This is perhaps the most frequently encountered thermal effect. Every winter brings its quota of frostbitten ears in certain geographic areas. Frostbite may occur as an incident in high altitude flying and constant vigilance is necessary to prevent its occurrence in aviators engaged in such work. Some cases result from careless or prolonged use of the ice cap.

The sequence of pathologic changes in the tissues affected by cold is not greatly different than that produced by heat and consists of engorgement of the blood vessels as thawing of the part takes place followed by stasis of blood in the capillaries, the exudation of serum and vesiculation. The degree of change in the tissues will be in direct proportion to the length of exposure to cold and the degree of temperature encountered. The victim of frostbite may be wholly unaware of its onset and suffer prolonged exposure because of lack of the warning of pain. In severe cases necrosis of tissue and loss of substance from sloughing will occur.

The first step in treatment consists in thawing the part and restoring normal circulation. This is best accomplished by gentle friction with the hand. It is generally believed that a frozen member should be thawed slowly by using snow or cold water applications but medical opinion does not support this belief. The specialist will be chiefly concerned with the after treatment of such conditions, the principles of which are essentially the same as those for the treatment of burns. The prevention of infection and the preservation of nourishment to the part are the chief objectives to be attained in treat-

ment. The application of bland ointments is of considerable value. The ointment should be applied on sterile cotton pads and fresh applications made daily. Phenol is not a safe ingredient in ointments used in the treatment of frostbite as gangrenous changes may be encouraged by its use. The relief of pain is best accomplished by internal medication with sedatives. Constitutional reactions are not common as the areas involved are usually small but if they do occur they are treated symptomatically. A satisfactory ointment for use in the initial stage of treatment is the following:

R ₁ Sulfanilamide	14 grains (0.9 gm.)
Sulfathiazole	14 grains (0.9 gm.)
Boroglyceride	1 drachm (4.0 cc.)
Lanolin	2 drachms (8.0 cc.)
White petrolatum to make	1 ounce (30.0 gm.)
Mix, and make an ointment	
Sg. Apply locally	

Burns (Dermatitis Ambustionis, Dermatitis Ab Igne).—Burns result from a great variety of causes in which heat acts on the tissues. The changes induced vary from simple hyperemia to marked destruction of tissue. The degree of change serves as the basis for classification of burns into three degrees. The principles governing the treatment of burns about the ear are the same as those for burns elsewhere in the body. Constitutional reactions may follow absorption of denatured protein producing temperature elevation, nausea, vomiting and in severe cases cerebral stimulation which may proceed to hallucinations and disorientation.

The chief danger of absorption is the production of degenerative changes in the liver and kidneys, particularly the latter. It is important to prevent absorption and avoid infection in the involved area. At the present time sulfonamides or tannic acid preparations are the favored local applications. Plain petrolatum is favored by some schools of thought. Regardless of what local application is used, particular care must be exercised to prevent the pinna from becoming adherent to the adjacent skin.

Sunburn (Dermatitis Solare).—Exposure to the sun's rays results in hyperemic reactions in the skin ranging in degree from mild to severe. Persons with fair skin are the most susceptible. Blondes will blister from sun effect while brunets tan. In some persons sunburn may follow exposure of short duration, twenty minutes exposure being sufficient in some instances to produce a marked skin reaction. The

effects of sunlight on the skin are heightened in the presence of snow, or on bodies of water, owing to the added effect of reflection from such surfaces. The changes induced by the sun's rays range from simple hyperemia of the exposed part to marked edema and vesiculation. Exposure of large areas of the body is distinctly dangerous and is quite often, if not always, accompanied by constitutional reactions.

Sportsmen, beach visitors, and others who anticipate exposure to the direct action of the sun's rays, especially city dwellers unaccustomed to such exposure, should take precautionary measures to prevent sunburn by the use of wide brimmed hats and local application of some substance to act as an obstruction to the passage of the sun's rays. Zinc oxide or calamine in either liquid petrolatum or ordinary lubricating oil such as that used in automobiles is quite effective as a preventive of sunburn, but it has its limitations beyond which it cannot be expected to work. Most applications are somewhat unsightly but this minor objection is more than compensated for by the discomfort prevented. A thick, creamy preparation may be obtained by mixing zinc oxide or calamine ointment with oil. The usual calamine lotion is too easily washed away. Zinc oxide or calamine powder as such have limited usefulness. Large areas of the skin may be safely anointed.

The same preparations are used for treatment as in prevention. Camphor alone or in combination with menthol may be added to the preparations for the cooling effect. A satisfactory lotion is

R ₁ Powdered camphor	10 grains (0.65 gm)
Menthol crystals	5 grains (0.325 gm)
Calamine powder	1½ drachms (6.00 cc)
Liquid petrolatum to make	3 ounces (100.00 cc)
Mix	

Sig. Apply locally

This must be well shaken before each application, and after it has served its purpose it is removed by washing with soap and water. The quantity of the ingredients may be varied to suit individual tastes. In severe sunburn with vesiculation the vesicles should be emptied and a bland ointment applied with special emphasis being placed on the prevention of infection for which purpose the ointment of sulfathiazole and sulfanilamide noted earlier in this section is quite satisfactory. A somewhat more elegant preparation may be made with sodium stearate

paste or a greasless cold cream base. Such a formula would be

R ₂ Camphor	2 drachms (8.00 cc)
Menthol	5 grains (0.325 gm)
Calamine	2 drachms (8.00 cc)
Zinc oxide	2 drachms (8.00 cc)
Greasless cold cream to make	3 ounces (100.00 gm)
Mix, and make a paste	

Sig. Apply locally

Radiant Energy Effects.—Ultraviolet light, roentgen rays, and radium are physical agents capable of inducing profound tissue changes, the effects varying from mild to severe, ranging from simple hyperemia to necrosis, depending upon individual susceptibility, length of exposure, and intensity of the rays.

Ultraviolet Light.—This agent is similar to sunlight in its effects, but it does not produce changes as marked as those induced by radiant energy of shorter wave lengths such as the roentgen ray and radium, but is quite capable of producing troublesome burns. Such burns are usually encountered in persons unfamiliar with the potent properties of ultraviolet light and are not infrequently encountered in patrons of gymnasia, health clubs, turkish baths, and other places where the ultraviolet lamp is used more or less indiscriminately and with little or no knowledge of its physical power. Exposure to the ultraviolet light must be for short periods of time in persons whose skin has not become tolerant to its effects by repeated exposure. Blondes require less ultraviolet light than brunets to induce a reaction in the skin. Short exposure to ultraviolet light produces simple hyperemia of the exposed part, which if carried a step further gives rise to exudation of serum into the tissues with swelling and edema. Vesiculation, bleb formation, and necrosis of tissue will follow if the exposure has been sufficiently long.

Roentgen Ray.—The extensive therapeutic use of this physical agent in present day practice makes a knowledge of its effects a practical necessity. In the treatment of many conditions of the external ear the roentgen ray is of great value. Mild exposure produces little noticeable reaction either subjectively or objectively. Prolonged single applications or too frequently repeated fractional treatments give rise to marked tissue reactions which may be alarming in degree. Moderate exposure produces simple hyperemic changes in the skin which may or

may not be accompanied by the sensation of burning. Larger dosage produces more intense effects, such as pigmentary changes in the skin, exfoliation of epithelium, and the production of scar tissue which subsequently contracts and leads to circulatory impairment and ulceration. More remotely, malignant degeneration may occur in the involved area. Persons of light skin pigmentation are especially susceptible to the roentgen ray and this should be borne in mind to avoid unpleasant after effects. Fractional doses of the roentgen ray frequently repeated, as in the Coutard technic, give rise to thickening and edema of the exposed skin with subsequent exfoliation of the superficial layer of epithelium and pigmentation. A single exposure of suf-



Fig. 210.—Lymphedema of the lobe of the ear and partial destruction of the tragus of the left ear in an elderly man due to epithelioma and radium treatment.

ficient intensity will produce a roentgen ray burn. The effect of exposure to the roentgen ray reaches its peak in about two weeks. The more marked reactions are characterized by hyperemia, edema, and thickening of skin through edema, exfoliation of epithelium, and even loss of substance. The subjective symptoms in roentgen ray burns are particularly annoying and will tax the ingenuity of the physician who is called upon to treat them. The burning sensation and formation are at times intense and unbearable to the patient. Relief may be difficult or impossible to obtain. In the healing process, cicatricial changes come on with atrophy of the skin and intensification of the subjective symptoms. Areas of telangiectasia appear and in some instances malignant degeneration occurs. The malignant changes may come on after months or years.

Prophylactic measures should be foremost in the thoughts of anyone using the roentgen ray either diagnostically or therapeutically. Prolonged or too frequent exposure to the roentgen ray is to be avoided. Correct filtration of the rays is also highly important in preventing adverse reactions. Because of the prompt and effective relief afforded in itching and painful conditions about the ear, it is very tempting to resort to repeated use of roentgen ray for relief and often before it is realized, an overdose has been given.

Radium—This chemical substance produces changes similar to but more pronounced than those produced by the roentgen ray. Radium is of great value in treating certain lesions about the ear, particularly malignancies, but its use should be entrusted only to those who are thoroughly familiar with its effects.

Contact Dermatitis—Inflammations of the skin due to contact with irritants or substances capable of acting as irritants under certain conditions are legion in number. However, there are a few more or less well demarcated groups into which the cases of contact dermatitis are classified.

Chemical Dermatitis—Simple dermatitis of the skin of the external auditory canal and pinna may be caused by contact with a great variety of chemicals. Under this heading are included purely chemical irritations not associated with hypersensitiveness or allergy. A typical example of such reaction would be the effect of trichloroacetic acid, lye, mercurial compounds, turpentine, or nitric acid on the skin. Many other substances have been designated as the cause of chemical dermatitis. Such substances are quite often used in treating lesions of the skin such as verruca. Acids and alkalis act somewhat similarly in the production of inflammation. It is noteworthy that the alkalis produce a more persistent type of local lesion than do acids. Chemical burns are often encountered in workers in industrial plants. The treatment of lesions of this nature consists in removing the irritant and neutralizing its effects as promptly as possible. Some of the chemicals used in industry are very active and destruction of tissue takes place very quickly following contact with them. Sulfuric acid, acetyl chloride, nitric acid, ammonia, and sodium and potassium hydroxide are only a few of the possible offenders that may be mentioned. The use of precautionary measures and safety devices is

imperative in the prevention of chemical burns in industry. Chemical dermatitis of lesser degree, such as would follow the treatment of local lesions with cauterants, usually responds promptly to the application of neutralizing agents. The chief concern in treatment is the prevention of destructive lesions from excessive chemical action, or extension into deeper tissues and protection from secondary infection. In medical practice the effects of chemical substances should be promptly neutralized as soon as they have served their purpose. Protection of adjacent skin with some type of ointment will often limit the extent of the effect of such agents. In industrial plants, neutralizing solutions should always be on hand and distributed in numerous easily accessible places, and in sufficient quantity to treat large areas if necessary, as prompt first-aid treatment produces the most satisfactory result.

Dermatitis Medicamentosa—Dermatitis medicamentosa comprises that form of skin irritation due to the local action of drugs used therapeutically. A distinction is made between chemical dermatitis, dermatitis medicamentosa, dermatitis venenata, and allergic dermatitis, although a degree of overlapping of the types must necessarily occur. The term "idiosyncrasy" was formerly much used in medical writings and denoted hypersusceptibility to drug action as well as hypersensitiveness or allergy. Dermatitis medicamentosa and allergic dermatitis overlap and are separated from each other on the basis of hypersusceptibility on one hand and specific hypersensitiveness on the other.

It is well known that certain individuals are more susceptible than others to the effects of drugs applied to the skin. Blonds are more vulnerable in this respect than brunets, a practical point which should be borne in mind in prescribing local applications. Phenol and allied compounds, derivatives of cresylic acid, ammoniated mercury, sulfur, turpentine, and at times even the mildest, apparently innocuous substances may cause local reactions. If a local inflammatory lesion fails to respond in the customary manner to a local application, one should not hesitate to reduce the strength of the preparation or change to something else. Overtreatment may be the cause of unduly persistent local inflammations.

Dermatitis Venenata—Dermatitis venenata is an acute, at times fulminating, reaction in the skin due to susceptible persons coming in con-

tact with certain plants such as primrose (*Primula obconica*), poison ivy (*Rhus diversiloba*), and poison oak (*Rhus toxicodendron*). It is characterized by the occurrence of an inflammatory reaction in the skin with the formation of multiple thin walled vesicles and bullae which tend to rupture and become confluent. There is admittedly much overlapping of types and causes, but we have chosen to separate the types to facilitate discussion. In dermatitis venenata of plant origin there is a definite degree of hypersensitiveness or allergy on the patient's part. Many persons can be exposed to these plants with impunity while others manifest the severest reactions to exposure of small areas. The lesions are attributed to the action of an irritant oil elaborated by the plant which is easily transferred from one part of the body to another by the patient's hands. The pinna is a frequent site for such lesions to occur.

Injections of dilute toxin of the offending plant as a preseasonal treatment affords protection to most persons. The injections may be bolstered by the subsequent oral administration of plant extracts designed for the purpose. There are several firms marketing such extracts in individual packages.

In the presence of dermatitis venenata, local applications of alkaline solutions such as sodium bicarbonate serve to neutralize the irritant oil by converting it into a soap which can be washed away. An alkaline soap will serve equally well. Greasy applications dissolve the oil and facilitate its spread. Cleansing should be followed by drying the parts and the subsequent use of a sedative lotion. Weak copper sulfate solution and camphor water are highly favored local applications. Calamine lotion with or without phenol may be used. The following is a satisfactory solution which may be applied as a wet dressing.

℞ Copper sulfate	10 20 grains (0.75 1 325 gm.)
Camphor water	16 ounces (500.00 cc)
Distilled water	32 ounces (1000.00 cc)
Mix and make a solution	

Sig. Use as wet dressing

The hypodermic administration of the immunizing extracts has proven of great value in our hands in causing prompt subsidence of the lesions of dermatitis venenata of plant origin. It is administered in fractional doses over a three-day period. The immunizing extract is obtainable in a special calibrated syringe con-

tainer. Its use is recommended both prophylactically and therapeutically. It is available for both poison oak (*Rhus diversiloba*) and poison ivy (*Rhus toxicodendron*).

Blister-Bug Dermatitis—This is a fulminating lesion produced by a beetle, which is common in the southwestern part of the United States, known as the blister bug by the laity (the *Epicauta* and *Pomphopoea* say). Mere contact with this insect produces a prompt and painful vesicular or bullous lesion at the point of contact. No one seems to be immune. The lesions produced by the blister bug must at times be differentiated from those due to such irritants as poison oak and poison ivy. There is marked burning and discomfort at the site of the lesion. Treatment consists in the use of sedative lotions.

Allergic Otitis Externa.—This is a dermatitis characterized by hyperemia, inflammatory reactions, edema, and, in severe acute cases, vesiculation. In the more chronic types, eczematoid changes occur. Allergic dermatitis is more frequent than is generally supposed and is due to the effect of some irritant or allergin to which the patient is specifically hypersensitive.

The exudative or serous type of eczematoid dermatitis due to allergy is usually confined to the acute types, while, in the longer standing more chronic types, thickening (lichenification) and scaling (desquamation) of the skin are prone to occur. The latter condition is referred to as squamous, or hyperkeratotic, eczema. The changes may only represent different degrees of reaction in the skin of a hyperseesitive person, and one type may merge imperceptibly into the other.

Fur neck pieces or fur collar trimmings are a fairly frequent cause of reactions about the ear. Cosmetics may contain substances which will produce such reactions,orris root being the chief offender in this respect. Face powders, face creams, and lotions should all come under suspicion in such cases. Intermittent exposure to an irritant may give rise to a puzzling otitis externa which varies in severity from day to day. Fingernail polish has recently been indicted as the cause of otitis externa. Scalp lotions or medicaments used about the ear may produce such reactions. Perfumes, essential oils (oil of bergamot), phenol and cresylic acid and their compounds may be the offenders. The various sulfonamides so popular at the present time may cause allergic skin reactions. Many cases have been reported in recent months. Inasmuch

as many of the preparations used routinely in otologic practice can produce local reactions it is always well to bear this possibility in mind, and in any given case where there is an exacerbation of symptoms while the patient is under treatment, or failure to respond to treatment in the orthodox manner occurs, such treatment should be discontinued forthwith and a bland local application substituted, or applications should be stopped altogether to allow time for recovery to take place.

Intravenous calcium gluconate or levulinate in one gram doses daily (for an adult), and parathyroid extract administered hypodermically (usual adult dose 0.5 cc daily) and combined with calcium lactate, gluconate, or diphosphate and parathyroid gland extract orally are of value in the treatment in most cases. Dilute hydrochloric acid by mouth is often helpful in lessening the hypersensitive state. Dietary readjustment with the avoidance of starches and sweets is particularly important. Elimination diets may be tried in refractory cases or specific desensitization may be employed after ascertaining the allergin. Mild roentgen-ray therapy will be of value to lessen distress and in the serous or exudative types is especially valuable. Extreme caution should be exercised in its use in such cases and it must be borne in mind that this is in no sense a curative measure. The aid of an allergist may be necessary in the most difficult cases. An exact study of the patient's body chemistry is always in order, especially if the patient proves refractory to treatment.

Infections—The infections of the external ear commonly encountered are classifiable as non-pyogenic, pyogenic, and mycotic. They comprise a very important part of otologic practice as they are of frequent occurrence. Great satisfaction accrues to both patient and physician when such infections are properly handled and prompt results obtained, but slow results from ill-chosen treatment lead to dissatisfaction and loss of the patient's confidence. External ear infections are usually very uncomfortable and prompt relief is not always easy to obtain.

Impetigo Contagiosa—This is a common pyogenic infection of the skin attributed variously to the action of streptococci and staphylococci. It produces inflammatory lesions characterized by a separation and loss of the top layer of epithelium with the formation of denuded areas which are covered with bright

yellow crusts. The lesions of the external ear are not essentially different from similar lesions elsewhere in the body. This type of infection often invades the external auditory canal particularly in children. It may affect the external ears of adults as well as children, though it occurs more frequently in the early years of life. It may assume epidemic proportions in institutions and is more frequently encountered in warm weather at which season the skin is more vulnerable. It is more frequently encountered in persons whose standard of personal hygiene is low but even the most fastidious person may be the victim of the infection. The primary site of infection may be the pinna or external auditory canal and the infection may spread to contiguous skin areas. The more severe cases

antiseptic soap such as synol or neko is satisfactory for this purpose. Ammoniated mercury in 5 per cent ointment is a time honored effective local application. A stronger concentration may cause sufficient chemical irritation to perpetuate the lesions and the objective of treatment may be defeated entirely or the case prolonged unnecessarily. Starch added to the ointment base makes a paste which absorbs fluids and when this type of ointment is applied serous exudation is less likely to lift the medication away from the lesions. The sulfa drugs may be used locally in ointment form or in aqueous or oily suspension and their effect reinforced by internal administration of the same class of drugs. Penicillin ointment is a promising recent addition to the drugs used in combating



Fig. 211.—Impetigo contagiosa of the left external auditory canal in a Mexican child. The infection is extending to the skin surrounding the entrance to the canal.

are accompanied by constitutional reactions but in the average case this does not occur. Impetigo contagiosa responds quickly and favorably to proper medication. Healing takes place with no residual scarring although an area of hyperemia may persist at the site of the local lesion for several weeks.

In the treatment of impetigo one of the first requirements to be fulfilled is the prevention of extension of the infection to other persons. This calls for the exercise of the usual individual precautions, isolation may be necessary, especially in institutions such as orphanages. For local treatment to prove effective the agent must come in contact with the diseased area which necessitates removal of the yellow crusts. Cleansing with warm mercury bichloride or mercury cyanide solution (1:10,000) and an

pyogenic infections. An ointment containing 200 units of penicillin to 1 gm. (15 grains) of aquaphor provides an ointment which has given remarkably prompt results in a few cases of pyogenic infection of the skin such as impetigo contagiosa and pyogenic dermatitis.

Pyogenic Dermatitis (Dermatitis Pyogenica, Pyoderma, Coccogenic Dermatitis, Infectious Eczematoid Dermatitis).—In pyogenic dermatitis the lesions are due to the action of pyogenic organisms in which the invasion of the skin is deeper than in impetigo. All the pathologic changes are more marked than they are in impetigo as there is extension into the deeper layers of the skin and even into the underlying tissues, such as the cartilage of the ear, with loss of substance in severe cases.

Treatment is similar to that of impetigo.

After properly cleansing the lesions, an ointment of ammoniated mercury (5 per cent) or one containing sulfanilamide and sulfathiazole is applied. Internal administration of the sulfonamides in resistant cases is advised. Staphylococcus bacteriophage given hypodermically is often of value. Treatment with the roentgen ray is often beneficial in alleviating pain and discomfort and apparently assists in combating the infection and promoting healing. Its limitations should be thoroughly understood. It is of distinct value but can cause great harm. Prevention of auto-inoculation is important and is accomplished by bathing the skin in the surrounding area with some type of nonirritating antiseptic. A weak solution of mercury cyanide is satisfactory for this purpose.

Acute Abscess (Furuncle, Boil in the Ear, Bealing in the Ear, Pimple in the Ear)—This is an acute circumscribed pyogenic inflammation of the skin of the pinna or external auditory canal with the formation of one or more small abscesses in the involved area. The usual pyogenic organisms are encountered in these lesions—the staphylococcus, the colon bacillus, Friedländer's bacillus, and the Bacillus pyocyaneus. The lesions may be single or multiple and have a marked tendency to recurrence. The pain is out of all proportion to the magnitude of the lesions and is chiefly due to close adherence of the skin to the underlying sensitive perichondrium or periosteum. A well developed furunculosis of the external auditory canal causes profound distress and suffering. The swelling and pain may interfere with opening the jaws and may even cause the affection to be confused with parotitis. The lesions may extend into the deeper structures and produce perichondritis or chondritis, in which instance the inflammation may prove quite intractable and progress to the point of causing considerable deformity of the external ear by sloughing and loss of substance. Furuncles may dissect underneath the skin and perforate into the temporomandibular joint. Enlargement of the adjacent lymph nodes is the rule and these areas may become as important clinically as the initial focus of infection. Recurrent furunculosis of the external ear or canal calls for an investigation of the patient's general health, and there is likely to be an associated diabetes or metabolic disorder which would influence the course of such infections.

The opening of the swimming season heralds

the advent of furunculosis of the external auditory canal as the introduction of water into the external auditory canal lowers the vitality of the skin and makes it more vulnerable to infection. Considerable controversy often centers about this point. No doubt, there are instances where infection gains entrance by the introduction of contaminated water into the external canal, but equally often the organisms are already present in the canal and the skin merely made more susceptible by maceration. The investigations of Taylor^{1, 2} on the hygiene of swimming are most instructive and all persons interested in this subject are referred to the writings of this authority for further information.

Furunculosis is frequently encountered in otologic practice and, because of the pain accompanying it, demands prompt and energetic treatment for its relief. Quite often only partial relief from the pain can be obtained, and the patient should be informed of this fact as this condition often leads to dissatisfaction on the part of the patient and is not infrequently the cause of the patient changing doctors. Every otologist has his favorite prescription for the treatment of furuncles of the external auditory canal which he feels excels all others in therapeutic activity. A critical investigation of the various substances used in treatment will, however, reveal that many of them are relatively ineffective. McBurney and Searcy³ have investigated the bactericidal and fungicidal activity of a great many supposedly antiseptic substances commonly used in the ear and have found that many of them possess little effectiveness. The report of their investigations is worthy of review by all otologists.

The roentgen ray is often quite valuable in treating furunculosis in its incipency, as it is sedative and apparently limits to some degree further development of the infection. The use of staphylococcus bacteriophage hypodermically is of definite value in most patients. It is administered in the arm or leg. Direct injection of bacteriophage into the infected area is too painful to be of practical value and there is danger of disseminating the infection by pressure of the infiltrating fluid. Wet dressings using the bacteriophage may be employed, the results are indefinite as absorption of this agent through the skin is probably nil. The use of heat or cold applications often proves a valuable adjunct to treatment. Most patients prefer warm appli-

cations to cold, and moist dressings are preferable to dry. At the present time incision into the offending furuncle is rarely made, since the danger of spreading infection into the deeper tissues is increased by this maneuver and it is attended with considerable pain necessitating in most instances the use of general anesthesia. However, one should not hesitate to open a furuncle if it is already pointing and ready to rupture spontaneously.

The choice of local medication is important. The ideal drug is one which is anesthetic or analgesic and at the same time bactericidal. Metacresyl acetate (Sulzburger) marketed under the trade name of 'Cresatin' is the most satisfactory local application which we have encountered for this purpose. Gill⁴ has called attention to the value of this drug in the treatment of infections of the external ear. The technic advocated is as follows:

After cleansing the involved area by swabbing, aspiration or blowing a stream of compressed air into the canal through a fine metal cannula, a cotton wick saturated with Cresatin is inserted into the depths of the canal where it is allowed to remain twenty-four hours. When the external auditory canal is closed by swelling, only the smallest wisp of cotton can be introduced into it. Cotton wrapped on a wire applicator and saturated with the medication may be carefully inserted into the depths of the canal in such cases. It can then be stripped off the applicator and should be allowed to remain in situ. No attempt should be made to forcibly introduce a bulky wick into a canal which is the seat of such marked reaction. The wick is removed in twenty-four hours at which time further cleansing and treatment can be carried out with less discomfort. The wick is kept wet with the medicament by repeated applications with a medicine dropper without disturbing it. Cresatin and olive oil equal parts are usually employed for this purpose.

Gentian violet, mercurochrome, and neutral acriflavine and many other medicinal dyes have been used in the treatment of furunculosis of the external auditory canal. Vaccines—autogenous or stock—may prove of value in recurrent furunculosis. They are most effective in staphylococcal infections. Insulin in fractional doses is often of great value in combating such infections, particularly the recurrent types. The sulfa drugs serve well in furunculosis of the ear. They are used internally and also in ointment form. Wet dressings utilizing tyrothricin are worthy of trial in all pyogenic infections about the ear.

The following ointment is a valuable substitute for the older ammoniated mercury ointment

only in the treatment for furunculosis but also for impetigo contagiosa of the ear and pyogenic dermatitis.

R	Sulfanilamide	2 drachms (8.0 gm)
	Sulfathiazole	3 drachms (12.0 gm)
	Aquaphor	1½ ounces (45.0 gm)
	Petrolatum to make	3 ounces (100.0 gm)

Mix and make an ointment

Sig. Apply locally

Note. In preparing this ointment the sulfanilamide is mixed with the petrolatum. The sulfathiazole is dissolved in a minimum of water and mixed with the aquaphor. The two batches of ointment are then mixed together.

Antibiotics, such as penicillin, gramicidin, tyrothricin, and others, are at the present commanding great attention and may be said to occupy the center of the therapeutic stage at the moment in the treatment of pyogenic infections.



Fig. 212—Method of inserting cotton or lamb's wool wick into the external auditory canal. A bayonet shaped forceps is most satisfactory for this purpose. The cotton wick is saturated with the medication of choice and gently fed into the canal. It may be impossible to insert a wick in this manner if the canal is badly swollen and extremely painful.

Their use, while still in its infancy, gives great promise, and undoubtedly the medical profession will rely to a greater extent on these drugs as time passes and as they become more readily available. Every patient with a resistant or serious pyogenic infection should be given the benefit of the use of such drugs when they are applicable, but it should be borne in mind that they are only bacteriostatic in action, and also that they are not intended to supplant other well established methods of treatment. Cer-

tainly they will not supplant imperative surgical procedures, and should not be expected to do so. The antibiotics, such as penicillin, act synergistically with the sulfonamides, and the two classes of drugs may be used either supplemental to one another or coincidentally. These drugs because of their bacteriostatic properties (not bactericidal) may be considered as merely holding the line of defense until such time as the patient's defenses may be marshalled in force and brought to the scene of action for final extermination of the infection.

Penicillin is quite spectacular in its effect in certain infections but there are many resistant strains of bacteria that are either unaffected by it, or affected only in a negligible degree. It is



Fig. 213—Cellulitis acute, about the left ear in association with chronic discharging middle ear and acute cellulitis of the left external auditory canal. Note the profuse discharge evidenced by a drop of pus suspended from the lobe of the ear.

unwise to expect too much from penicillin and to abandon all the time-tried procedures in its favor. As a supplemental therapeutic agent it is of the greatest value.

Acute Cellulitis—Acute cellulitis of the external auditory canal may follow minute abrasions or breaks in the integument which permit the streptococcus to gain entrance. It may follow such an insignificant thing as an insect bite or a simple abrasion of the skin of the external auditory canal from the not uncommon practice of scratching it with a foreign body, such as a hairpin or metal paper clip. The treatment is essentially the same as that outlined for furunculosis.

Acute Perichondritis—Inflammatory reactions in the perichondrium of the ear may rep-

resent an extension of a superficial infection, such as an impetigo or a furuncle, or occur as a sequela to an injury such as an infected punctured or incised wound. Bites or stings of insects may be responsible for the perichondritis. Frosthite accounts for a sizeable percentage of the cases. Undoubtedly, some of the lesions are of hematogenous origin. In instances in which the etiologic factor is not apparent, serologic studies should be made since syphilis is an occasional cause, particularly in lesions of a diffuse and persistent nature.

Invasion of the cartilage by infection, or the jeopardizing of its nourishment by separation of the perichondrium through subperichondrial effusion, may lead to disintegration and loss of cartilage, and a shrunken, deformed pinna resembling the cauliflower ear of prize fighters will result.

Treatment is essentially that of the underlying cause. When infection is responsible the newer drugs of the sulfa class may be utilized both externally and internally. The evacuation of serous or purulent exudate is imperative and must be accomplished aseptically. Heat may be judiciously employed to stimulate circulation. Limitation of exudation by the hypodermic administration of parathyroid extract and the intravenous exhibition of calcium gluconate or levulinate is advisable. In syphilitic cases the orthodox methods of treatment are used.

Erysipelas—Erysipelas is an acute inflammatory disease of the skin of unusual severity which is due to the action of a specific strain of streptococcus. It may occur spontaneously or follow operations such as those on the mastoid. It is easily transmitted from one person to another and may occur in epidemic form in institutions. It is accompanied by marked constitutional reactions, high temperature, and is at times fatal. The pathologic picture is that of an acute capillary lymphangitis and it is the lymphatic vessels in the involved area which bear the brunt of the attack. For this reason the involved area is sharply demarcated.

Prophylaxis consists in isolation of known cases of erysipelas and the observance of individual precautions to prevent spread of the infection. When operation is contemplated in known streptococcal cases, the internal administration of the sulfa drugs preoperatively will diminish susceptibility to erysipelas.

The sulfonamide drugs have provided us with a powerful weapon for the treatment of infec-

tions of this class, and while there is some difference of opinion expressed in current medical literature concerning the degree of effectiveness of these drugs in the treatment of erysipelas it is believed that, in the light of our present knowledge, they should be given a trial in every case. I have found the erysipelas serum manufactured under special license by the University of Rochester, New York, to be particularly valuable. It acts promptly and effectively, but it must be repeated several times in the average case. Urticarial reactions are frequent following its use, but they are promptly controlled by the hypodermic administration of

with ichthyol in alcohol, may be used. It is doubtful if local applications influence the course of the disease materially, but they may relieve to some degree the burning sensation which attends such lesions and thus contribute greatly to the patient's comfort.

Otitis Externa Mycotica (*Otomycosis*, *Fungus Ear*, *Swimming Ear*, *Panama Ear*, *Tropical Otitis*, *Mildew Ear*, *Singapore Ear*, *Adobe Ear*) — Mycotic otitis externa is that type of inflammation of the external ear (pinna and canal or both) in which the causative organisms are molds. Three types of molds are encountered in such cases: (1) budding forms, (2) fila-

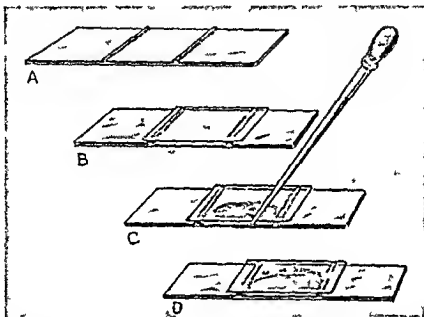


Fig 214—A method of making slide culture for fungi. A, Strips of melted sealing wax or dental modeling compound applied to slide. B, Coverglass is placed in position while the wax is hot. C, Material to be grown is mixed with melted culture medium and introduced with a pipet into the space between the slide and coverglass, filling approximately one half of the available space. D, After incubation in a moist chamber (Petri dish containing a small amount of water) growth takes place at the free edge which can be examined microscopically.

adrenalin. Penicillin administered intravenously may prove to be our most powerful weapon in erysipelas, but its effects have not been observed in a sufficient number of cases to warrant an expression of opinion on its effectiveness at the present time.

A great variety of local applications have been used in treating erysipelas. Magnesium sulfate, normal saline solution, solution of boric acid, and camphor water compresses are a few that may be mentioned. Various mixtures have been painted on the involved area. Solutions of ichthyol or ichthyol in combination with glycerin or in flexible collodion may be beneficial. Tannic acid, alone or in combination

mentous forms, and (3) higher bacterial forms.

Budding forms are divided into *Saccharomycetes*, *Torulae*, *Monilia*, *Oidia*, and *Coccidioides*.

Saccharomycetes, or common yeasts, are ordinarily nonpathogenic but may become potent as disease producers under certain conditions (facultative pathogens). They are frequently the cause of low-grade dermatitis of the external ear or in the postauricular area.

Torulae are a rare budding type, sometimes referred to as wild yeast. They produce lesions which are potentially serious because of the visceral lesions which may be associated with

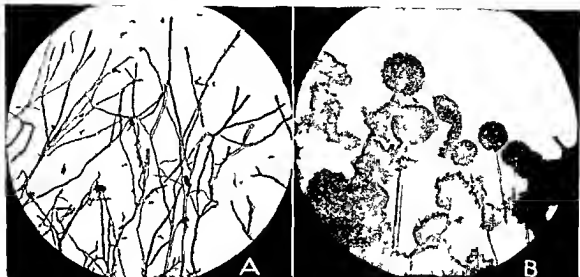


Fig 215—Photomicrograph of molds isolated from patients with otomycosis A The penicillium B the aspergillus Further classification of such molds is made by observing the cultural reactions on various media A slide culture is necessary for morphologic study A simple stained smear is inadequate for this purpose

the cutaneous manifestations of the disease They are rarely encountered in the ear

Monilia consists of a number of strains The organisms have been considered by many authorities as responsible for or in some way



Fig 216—Torulosis of the tragus in a middle Mexican woman where on the body numerous lesions isolated from each like

intimately associated occur frequently as in the southwestern States

The *Oidia*, of which typical representative, which may prove as

unless the true nature of the underlying organism is recognized They are the organisms which produce the thrush of older writers They have been reported as the cause of otitis externa

Coccidioides is a genus of budding organisms resembling the yeasts but not a true yeast It is classified with the yeasts for the lack of a better place It produces cutaneous as well as visceral lesions and is endemic in certain western states

Under the heading filamentous forms of molds there are only three commonly encountered types of organisms *Aspergilli*, *Penicillia*, and the *Mucors*

The *Aspergilli* comprise a large family with many members and many strains They are widely distributed in nature and are frequently encountered in mycotic ear infections In growing they produce a white, cotton like mass of which will be covered with blackening degrees when the stage of decay is reached for the spores of this mold are black and resemble fine

another of the filamentous molds produce otomycosis There are many in this family Coccidioides is one of them the most common of the ear molds in the tropics and subtropics They are often associated with the ear in the tropics and subtropics They are often associated with the ear in the tropics and subtropics They are often associated with the ear in the tropics and subtropics

they tend to disappear after a short time in mixed infections of the external auditory canal

The *Mucors* produce lesions similar to those attributable to the *Aspergilli* and *Penicillia*, with the formation of debris in the external auditory canal that is soggy and resembles putty in appearance. They are less frequently encountered in otomycosis than the other filamentous forms

The organisms coming under the heading *higher bacterial forms* possess properties which cause them to resemble both the fission fungi or bacteria (Schizomycetes) and the molds (Eumycetes). There is much confusion surrounding their naming as well as their classification. In consulting the literature on this subject one is immediately impressed with the synonymy of names. Thus the *Actinomyces* is also designated as *Streptothrix*, *Leptothrix*, *Cladothrix*, *Oospora*, *Nocardia* and *Discomyces*. The organisms may be mutation forms of a common ancestral type. Some are acid fast, others not, and this characteristic is used as a further basis for classification. Usually the group name is used to designate lesions caused by one of the strains coming under the heading of Actinomyces, a practise which would lead to clarification of the medical literature on the subject if universally adhered to.

Actinomycosis or *lumpy jaw* is an essentially chronic infection with any one of several strains of the *Actinomyces* or ray fungus. The disease affects both man and animals. The lesions are deep seated and the infection is characterized by its burrowing tendency and the formation of multiple foci of infection which progress to abscess and fistula formation. The exudate from the fistulas contains granules in which the *Actinomyces* are found. The granules are usually though not always bright yellow. The organisms composing them may be demonstrated by crushing a granule on a cover slip or between glass slides. The large organisms are easily demonstrated by staining with any of the simple bacterial stains. Systemic invasion with visceral involvement is practically 100 per cent fatal. The primary lesions of this disease may occur on the external ear or in the external auditory canal or these structures may become secondarily involved by extension of the infection from a nearby focus. Diagnosis is established by finding the fungus in the granules cast off in the discharge from the fistulas

by biopsy, animal inoculation or culture on artificial media.

Molds are widely distributed in nature and infection takes place easily in susceptible persons. Reinfection after apparent cure is not uncommon. The life cycle of the average mold encountered in the ear may be reckoned as two weeks, and flare ups may be anticipated at such intervals. Swimming is held responsible for infection in many cases. Probably the only relationship in most instances is the supplying of the necessary moisture that enables molds to grow prolifically or the lowering of skin resistance through maceration thus providing a better medium for mold growth.

Molds grow in the upper layers of epithelium and their growth produces itching and discomfort in the ear. The local reaction may at times become a severe atopic reaction if the patient is extremely susceptible to a given mold. Vesiculation and ulceration may take place. The infection rarely extends to the cartilage of the ear. Perforation of the ear drum may occur but is infrequent. Molds may be mixed with pyogenic organisms in chronically infected ears, but the pyogens tend to crowd out the molds. This occurs even with certain of the *Penicillia*, but probably does not occur in association with the *Penicillium notatum* from which penicillin is derived.

The sequence of pathologic changes produced by molds in the external ear is as follows:

1. Implantation of the organisms in the external auditory canal takes place.

2. Growth of the organisms follows—the rate depending on conditions of temperature, moisture or pre-existing irritation.

3. Invasion of epithelium occurs with attendant itching and discomfort which may be quite severe.

4. Exfoliation of epithelium ensues as nature attempts to overcome the infection by casting off the uppermost cells.

5. Denudation occurs from exfoliation as the top layers of epithelium are cast off and the canal becomes filled with debris.

6. Superficial ulceration and eczematoid dermatitis result if the pathologic process goes far enough. The changes do not always proceed through the entire sequence. Sometimes the molds produce changes of the mildest imaginable character which may be overlooked.

The molds, both filamentous and budding types, may be demonstrated in the material

from the infected ear by the method of Whalen⁵ which consists in the examination of a slide smear which has been treated with a solution of sodium sulfite (2 per cent) to which methylene blue has been added but exact classification of the causative organism is possible only by cultural and fermentation reactions. Slide cultures as well as Petri dish cultures on special media (Sahouraud's) should be made for exact study and classification. Intradermal injection of filtrates from responsible organisms assists in establishing the diagnosis in obscure cases.

In the treatment the use of alcohol medicated or plain in the ears after swimming is a good prophylactic measure. Mercury cyanide (1:5000) in ethyl alcohol (70 per cent) is a satisfactory liquid to use. Ear stoppers do no good unless their use is followed by some antiseptic in the canal.

The Pseudomonas pyocyaneus produces a resistant type of infection which responds to acetic acid or substances containing the acetate radicle such as a solution of aluminum acetate or a 2 per cent aqueous solution of acetic acid, or metacresyl acetate (Cresatin).

The treatment of patients with actinomycosis is both local and systemic. Iodine or the iodides administered orally and intravenously are the most efficacious drugs to use. An isolated lesion if discovered sufficiently early may be eradicated by the use of the actual cautery. Vaccines have been used with indifferent results. Copper salts particularly colloidal copper have been recommended but their value is doubtful. The actual cautery when applicable to the lesion is the most effective agent in the local treatment.

The treatment for otomycosis in general consists of mechanically cleansing to remove the epithelial debris either by swabbing or use of aspiration or cautiously blowing a stream of air into the canal through a petrolatum on the swab used. Ear lesions the burning of metacresyl acetate is to be resorted to in subsequent treatment. If furuncles picture extreme gentleness in cleansing may be observed or cleansing may be continued until local sensitiveness is usually accomplished within hours.

After cleansing I have found acetate to be a most satisfactory next step of the treatment. In

sons however it will produce local chemical irritation. The bactericidal and fungicidal properties of the drug are slightly enhanced by the addition of thymol but in some patients this produces an uncomfortable burning sensation. The metacresyl acetate is introduced into the external auditory canal on a cotton wick and is allowed to remain twenty-four hours. The treatment is precisely the same as that used in furuncles. After twenty-four hours the cotton wick is removed at which time the canal can usually be cleansed with little discomfort as the medication is also anesthetic. The epithelium of the canal will be white from contact with the drug; the top layer will be detached and this epithelial debris can be easily wiped away. The wick is reinserted and wet with metacresyl acetate. The patient is instructed to keep it moist with the following preparation:

R	Oil of cloves	3 minims (0.2 cc.)
	Cresatin	2 drachms (8.0 cc.)
	Oil of olive to make	1 ounce (15.0 cc.)
	Mix	

Sg. Apply as directed to cotton wick in the ear.

This treatment is employed for three or four days in succession and then a bland application is substituted. Ichthyol ointment is of value at this stage or the following preparation serves well if serous exudation has taken place:

R	Cade oil	15 minims (1.0 cc.)
	Oil of olive to make	1 ounce (30.0 cc.)
	Mix	

Sg. Apply to ear several times daily.

A more prolonged application is

Cade oil	15 minims (1.0 cc.)
assar's paste to make	1 ounce (30.0 cc.)
x, and make an ointment	
ly locally	

It in combination is also an excellent metacresyl acetate powder^{6, 7, 8} and trichresol (1 per cent) especially come for burning.

given or if a liquid is preferred the following is satisfactory

R₁ Tincture of deodorized opium 2 drachms (8.0 cc)
 Aromatic elixir to make 4 ounces (120.0 cc)
 Mix
 Sig Teaspoonful every two or three hours

Heat externally is gratifying to most patients but cold may be preferable as an inhibitor of bacterial growth

Staphylococcus bacteriophage or *staphylococcus toxoid* may be of use in the treatment of otomycosis associated with secondary pyogenic infection Vaccines—stock or autogenous—are effective in a fair percentage of recurrent cases The stock vaccines appear to be as efficacious as the autogenous The sulfonamides have provided the physician with an additional weapon

The coincidental use of iodides internally and mercurial preparations externally (or the reverse) should be avoided since a very severe chemical reaction will follow when these two drugs meet in the skin A similar reaction may follow when bromides have been used for sedation and subsequently mercurials are used in the canal or on the pinna Any drug may perpetuate an inflammation through its local irritant qualities

The work of McBurney and Searcy³ is recommended for serious study by all otologists who would acquaint themselves with the exact degree of bactericidal and fungicidal effect of the preparations commonly used in treating pyogenic and mycotic lesions of the external ear

Lesions Due to Infectious Diseases—*Tuberculosis of the External Ear (Lupus Vulgaris Tuberculosis Cutis)*—Tuberculous dermatitis may arise from direct inoculation of the tubercle bacillus into the skin of the pinna or external auditory canal and produce the so-called tuberculous chancre or primary complex A chronically discharging tuberculous middle ear or mastoid may be the origin of such a lesion in the external auditory canal or of the pinna Also tuberculous lesions may arise from internal foci these constitute the hematogenous or endogenous group Many morphologic variations of tuberculous dermatitis are described but regardless of the names designating such lesions it is important to remember that the disease is one and the same thing regardless of name Tuberculosis verrucosa cutis designates a warty overgrowth or excrescence at the point of inoculation which may spread

laterally or into the deeper tissues Lupus vulgaris is the type of tuberculous lesion in which tubercles occur in the top layer of the skin The lesions extend peripherally and coalesce with other areas They eventually break down but subsequently heal with fibrosis The latter may cause considerable distortion of the tissues and great discomfort to the patient

The lesions of cutaneous tuberculosis must be differentiated from those of leprosy lupus erythematosus cancer, syphilis mycotic skin diseases and occasionally from pyogenic lesions of the skin which have become subacute or chronic The differential diagnosis ordinarily presents no particular difficulty if the usual diagnostic measures are used such as culture animal inoculation examination of curettings and histologic examination of excised tissue

Tuberculosis of the skin usually has its onset before the affected individual has reached the age of ten years The lesions usually progress slowly The disease is chronic destructive and deforming Tubercles may be demonstrated in the so-called apple jelly nodules in the skin The lesions may be very painful Invasion of deeper tissues or constitutional involvement may occur by extension from a primary focus in the ear

The treatment is discouraging as little can be done to influence the outcome in any case When a circumscribed area is involved such as the lobe of the ear and the surrounding tissues are free from infection amputation of the affected part with the actual cautery is indicated as the disease may thus be eradicated and much subsequent suffering prevented Generally speaking however the treatment of tuberculous lesions of the ear is constitutional plus the use of heliotherapy locally Tuberculin therapy also has its ardent supporters For a more detailed discussion of the lesions of cutaneous tuberculosis and their treatment one is referred to the standard textbooks on dermatology

Syphilis of the External Ear—The lesions of syphilis affect the external ear the same as other tissues of the body When the primary lesion occurs on the ear the lobule is the most likely location because it is often subjected to infection through kissing The presence of an ulcer in association with an enlarged adjacent lymph node is strongly suspicious of a syphilitic chancre In the secondary stage of syphilis the macular and papular lesions occur which may be very easily overlooked or considered as due

to some other condition. Darkfield examination of the primary lesion and serologic tests will establish the correct diagnosis. Perichondritis and chondritis may occur and give rise to diagnostic difficulties. Application of proper remedial measures will cause prompt subsidence of the lesions.

Leprosy—This disease occurs in three forms: the maculo-anesthetic, the nodular, and the mixed forms. It is not as rare as is generally supposed. The exact mode of infection with the lepra bacillus is unknown, but the disease apparently has a long incubation period (years),



Fig. 217—Acute ulcer of the posterior aspect of the pinna with adjacent lymphadenopathy in a white woman thirty years of age. The ulcer was extremely painful, and there were moderately severe constitutional symptoms. The diagnosis of malta fever was made on this patient and the ulcer on the ear was thought to be related to the generalized infection. Serologic and dark field examinations showed no evidence of syphilis.

and prolonged association with an infected person appears to be essential to its spread. A persistent area of hyperemia in which there is persistent anesthesia may be the first sign of the disease. Nodules in the lobe of the ear are almost sure to occur in some stage of the disease. They may be the earliest sign encountered in leprosy. Diagnosis is established by biopsy and finding the lepra bacillus in the tissues.

Treatment in institutions devoted exclusively to the care of such infections offers the best chance of recovery. The employment of chaulmoogra oil and its esters has apparently re-

sulted in many remissions and quite a few clinical cures. The prolonged use of typhoid vaccine and the internal administration of thymol, in addition to chaulmoogra oil or chaulmoogra oil ester therapy, are suggested as worthy of trial.

Diphtheria of the External Ear—Infection of the skin of the auditory canal and pinna with *Corynebacterium diphtheriae* (*Bacillus diphtheriae*) is occasionally encountered. The organisms may be implanted directly on the skin and produce lesions at the site of inoculation, or, as is more often the case, reach the external auditory canal from the nose and throat, by way of a discharging middle ear.

Organisms morphologically identical with the diphtheria bacillus, but lacking the pathogenic attributes of the true diphtheria bacillus, are not infrequently encountered in otitis externa. These usually harmless diphtheroids may cause considerable apprehension until their true nature is revealed. The stained smear will often give a clue as to the classification of such organisms, as the tinctorial characteristics of the diphtheroids and the true *Corynebacterium diphtheriae* are different. For the purpose of differential staining, Alberts' stain is used. The polar bodies, as well as the bars characteristic of the pathogenic diphtheria bacillus, are thus revealed. Determination of the virulence of an organism at times becomes a very serious matter, for an ear that is the seat of an otitis externa due to virulent diphtheria bacillus can act as a focus from which the infection is spread. The organisms may be present in the ear after the nose and throat have cleared up and may persist for long periods of time, until recognized and properly treated. Animal inoculation (guinea pig) should be resorted to in doubtful cases, as well as culture of the suspected organism in the several sugar media. Culture on Löffler's blood serum medium alone is not sufficient for differential diagnosis. It should be borne in mind, however, that there is a potential danger present in diphtheroids, as they have been known to assume pathogenic properties on repeated or successive animal inoculations. The true diphtheria bacillus is fatal to guinea pigs with the production of diagnostic hemorrhages in the adrenal glands.

The type of lesion produced in the skin of the auditory canal or pinna may be the orthodox fibrinous, or diphtheritic membrane, but more frequently this is lacking. There may be only

diffuse inflammatory reaction in the skin, which resists ordinary treatment. The bacillus of diphtheria elaborates an exotoxin which possesses marked necrotizing properties, and there may occur, as the result of its action, a variety of lesions ranging from vesicular to ulcerative. Gangrene of the part may occur. In the longer standing cases, granular overgrowth of the tissues may result. In association with the lesions, constitutional reactions may be present. The local lesions may be fulminating in character and death may occur from the effect of absorbed toxin on vital organs, such as the heart.

In the acute infections, response to diphtheria antitoxin, administered parenterally, is

with scar formation which is permanent. There are marked constitutional symptoms. Occasionally the disease extends by way of the eustachian tube to the middle ear and thence to the external auditory canal.

Treatment of external ear complications consists of procedures to promote cleanliness and the use of bland ointments or lotions. The treatment of the ear lesions is essentially the same as that of the generalized manifestations of the disease. Otitis externa associated with this condition may require special attention and sedative local applications.

Varioloid—This is an attenuated form of smallpox occurring in persons previously immunized against smallpox. The disease is not as



Fig. 218.—Lesions of chickenpox (varicella) on the right pinna of a ten year old girl. Note the pleomorphic character of the lesions evidenced by the presence of macule, papule, pustule, and crusting lesion. Owing to secondary infection, the lesion on the lobe of the right ear is assuming the characteristics of impetigo.

usually prompt. Additional effect may be obtained by use of the serum locally. In the treatment of both the true diphtheria infections and the nonpathogenic diphtheroids, the local application of gentian violet has been extolled because of the selective action of this antiseptic for organisms of this type. The medicament is ordinarily used in a 2 to 5 per cent solution in 50 to 60 per cent ethyl alcohol. Exuberant granular masses are best treated with the actual cautery.

Varola (Smallpox)—The lesions of smallpox occur on the ear as well as on other parts of the body. Lesions on the ear have the same characteristics as those of the generalized eruption. The typical lesion is a pustule—usually umbilicated—which resolves by crusting. It heals

severe as smallpox, but has the same general characteristics. The complications about the ear are also the same. Treatment is essentially that outlined under variola.

Vaccinia (Vaccination, Vaccina, Vaccinola, Vaccinoid, Vaccinella, Cowpox)—The lesions of vaccinia may be local or general. Those occurring on the external ear are usually the result of auto inoculation from a vaccination on the arm. They have the same characteristics and proceed through the same developmental stages as they do on the arm or elsewhere on the body, and when they subside a permanent scar will remain. The present day custom of not covering the original site of inoculation on the arm with a shield or protective dressing would reasonably be expected to increase the incidence of

lesions by auto inoculation, but such does not appear to be the case in actual practice. Occasionally vaccinia lesions result from virus transferred from another person. Rarely a generalized eruption may follow vaccination and some of the vaccinia lesions may occur on the pinna or in the external auditory canal.

Treatment of vaccinia consists in opening the pustules or removing crusts and the application of 70 per cent alcohol dressings which are quite sufficient to destroy the virus and arrest the progress of the lesion.

Varicella (Chickenpox)—Varicella is an acute infectious disease, common in children, characterized by the occurrence of an eruption on the skin. The lesions come in crops, at intervals of twenty-four hours or more, and one may see at the same time lesions which are papular, pustular, and crusting. This feature is of diagnostic importance as it is characteristic of the disease. The early lesions undergo involution while new ones are forming. The disease runs a mild course usually subsiding in a week. The lesions of chickenpox are frequently encountered on the pinna or in the external auditory canal. They are ordinarily not especially painful, and usually heal with little or no scarring.

The disease is self-limited and usually no treatment is required for the lesions. The use of a bland ointment or calamine lotion may be necessary in some cases.

Seborrheic Dermatitis (Dandruff)—The lesions of seborrheic dermatitis consist of greasy scales which become heaped up, giving rise to thick accumulation of epithelial debris which is easily wiped away. The base upon which this occurs shows various degrees of inflammation, usually mild in degree. On the scalp the epithelium is piled up and exfoliated in the form of flat silvery scales known as dandruff. The condition is transmissible from one person to another. It is variously ascribed to the action of the *Morococcus* of Unna, or the *Pityrosporum* of Melendez. It may be spread by using hair brushes, combs, or similar objects in common with other persons, or by the use of tonsorial equipment which has been inadequately sterilized. The lesions are usually indolent in character and are almost always associated with considerable itching.

In treating seborrheic dermatitis of the ear, it is well to bear in mind that it will recur promptly if the scalp is infected and allowed to

remain so. The usual local treatment consists in applications of an ointment to the ear lesion. Salicylic acid, ammoniated mercury, sulfur preparations, thymol, benzoic acid, and a host of other drugs have been used. Salicylic acid is an essential ingredient of all ointments for seborrheic lesions. It may be necessary to combine antipruritic substances, such as menthol and camphor (alone or in combination), with the ointment. A good prescription is

\mathcal{R} Menthol crystals	2 grains (0.13 gm.)
Powdered camphor	10 grains (0.65 gm.)
Salicylic acid	5 grains (0.325 gm.)
Precipitated sulfur	1 drachm (4.00 gm.)
White petrolatum to make	1 ounce (30.00 gm.)
Mix, and make an ointment	

Sig Apply to the ear at least twice daily.

Caution must be exercised in not getting the local application too strong for it may thus add an additional irritation which would delay healing. Salicylic acid is keratolytic and thins the top layer of skin by its action. Once it has served its purpose, it should be discontinued and another type of ointment used. The formula just given, minus the salicylic acid, may be used after the scaling has subsided. Another satisfactory ointment is

\mathcal{R} Cade oil	20 minims (1.325 cc.)
Precipitated sulfur	1 drachm (4.00 gm.)
White petrolatum to make	1 ounce (30.00 gm.)
Mix, and make an ointment	

Sig Apply to the ear twice daily.

If the lesions are irritated by the ointment used in the initial phase of treatment it should be discontinued and a bland nonirritant ointment substituted. A satisfactory preparation is

\mathcal{R} Boroglyceride	1 drachm (4.00 gm.)
Lanolin	1 drachm (4.00 gm.)
White petrolatum to make	1 ounce (30.00 gm.)
Mix, and make an ointment	

Sig Apply locally.

Treatment to the scalp must be carried out simultaneously or the ear lesions will promptly recur when their treatment is stopped. The following preparation has proven to be satisfactory for treatment of the scalp.

\mathcal{R} Salicylic acid	10-15 grains (0.65-1.00 gm.)
Ammoniated mercury ointment 10%	1½ ounces (46.00 gm.)
Olive oil or Cottonseed oil to make	8 ounces (240.00 gm.)
Mix	

Sig Application for the scalp.

It is applied to the scalp at night and allowed to remain overnight, the head being wrapped in a band towel, Hindu style. Next morning it is shampooed away with an antiseptic soap, such as synol or tincture of green soap. A slightly alkaline soap serves better than a neutral one, therefore soft soap (*sapo mollis*) or tincture of green soap (*tinctura sapo viridis*) is desirable because of its slightly keratolytic action which aids in dissolving and loosening the dandruff scales. Repeated applications of the scalp ointment may be necessary, but treatments should be at least three days apart. Reinfection should be guarded against by the use of an antiseptic lotion particularly after visiting barber shops or beauty parlors where the standard of sterility of equipment may be low.

Eczema—Eczema may be classified as (1) exudative (sometimes called serous) eczema and (2) nonexudative, squamous or hyperkeratotic eczema. It is most difficult to draw the line separating chemical dermatitis, dermatitis medicamentosa, allergic dermatitis, and the various types of eczema one from the other. Eczema as a diagnostic term is being used less frequently as our knowledge of allergy and body chemistry increases. Quite often eczematoid lesions will disappear when dietary changes are made. The persistence of such lesions may hinge on the use of such simple foods in the diet as eggs, chocolate, wheat flour, the various starches, and the disaccharides particularly. These are only a few of the offenders that may be mentioned, but one should always be on the alert to discover any factors that may influence the course of skin lesions that tend to become chronic. Skin tests to reveal hypersensitiveness to various substances that may act as allergens and exclusion diets are often of value in detecting the exact substance that is the offender.

The treatment of eczema of all types resolves itself into the following: elimination of the irritant responsible for the lesion if it can be discovered, correction of deficiencies in diet and vitamin intake, and the use of such local applications as are necessary to aid in correcting the changes which have taken place in the skin.

Care should be exercised in the choice of local applications in the treatment of eczematous lesions for the drug that is intended to clear up a local lesion may, through its irritant properties, serve to perpetuate it.

Exudative Eczema—A persistent inflammatory reaction in the skin with loss of the covering epithelium, often associated with vesiculation or papule formation and the exudation of serum from the involved area, constitutes the clinical picture of exudative or serous eczema. Eczematoid lesions may follow the use of certain drugs locally in too great concentration or for too long a period of time. If and when secondary infection complicates the picture, the lesions are referred to as infectious eczematoid dermatitis.

Ointments containing the so called reducing agents are of value in the treatment of exudative lesions. Oil of cade or coal tar is usually employed for this purpose. There is a great variation in the kinds of tar available for prescription use, some of them are not altogether satisfactory. A typical preparation for use in the treatment of exudative eczema is

Rj	Cade oil	1-2 drachms (4.00-8.00 gm)
	Zinc oxide ointment—	
	to make	1 ounce (30.00 gm)
	Mix and make an ointment	
Sig	To be applied to the ear twice daily	

If there is an associated pruritus, menthol and camphor may be added to the prescription. Phenol is often of value, but produces a local reaction in a certain percentage (as much as 25 per cent) of persons. The roentgen ray is of value in controlling itching but must be cautiously employed. The internal administration of sedatives may be necessary to obtain relief and enable the patient to sleep. Calcium administered by mouth or intravenously is often of great value. Parathyroid extract (0.5 cc for an average adult patient), given hypodermically once daily, quite often gives surprisingly good results. In combination with calcium the action of the latter is increased.

Nonexudative, Squamous, or Hyperkeratotic Eczema—In this type of eczema the skin is thickened and the top layer is built up in varying amounts, giving rise to more or less scaly, dry lesions. Fissuring of such lesions is common and they may become quite painful. The causes are essentially the same as those for serous or exudative eczema except that the factors responsible have been acting over a longer period of time.

In the treatment, it is first necessary to reduce the thickness of the overlying skin by the use of some one of the keratolytics and for this pur-

pose salicylic acid is the favorite drug. It is preferably used in ointment form in strengths varying from 1 to 3 per cent. The following preparation is suggested:

R ₁ Salicylic acid	5 grains (0.325 gm)
Cold cream to make	1 ounce (30.00 gm)
Mix, and make an ointment	
Sig. For local application	

Salicylic acid may also be used in solution, preferably in ethyl alcohol (95 per cent), and in otologic practice this liquid medicament is quite a favorite.

Vitamin Deficiency in Otitis Externa—Within recent years the important place that the vitamins play in metabolism has become more thoroughly understood and the relation ship that deficiencies, slight or marked, bear to certain dermatoses has become more evident. The squamous type of dermatitis associated with pellagra, a well established vitamin deficiency, may be mentioned as a typical instance of the influence of vitamins on the texture of the skin. Hyperkeratosis of the skin of the pinna is regarded by many as positive evidence of vitamin deficiency. No doubt, many other dermatoses are dependent upon a deficiency of one or more vitamins for their occurrence.

Prophylaxis is self evident and consists in dietary readjustment to insure proper vitamin intake, supplemented by the administration of vitamins. Dosing with vitamins is not to be recommended as a substitute for an adequate diet, a practice which has become entirely too prevalent in the present age.

Herpes Simplex (Fever Blisters, Cold Sores)

—This disease, which is due to a virus, is characterized by thin walled vesicles on a mildly inflammatory base. In the ear the lesions usually occur on the lobule or pinna but are also fairly frequent in the external auditory canal. The lesions are self limited. The vesicles tend to rupture spontaneously and coalesce causing a denuded area from which serum exudes. During the stage of regression the lesions are covered with a yellowish brown crust which detaches spontaneously, when healing is complete leaving a more or less conspicuous reddish area which clears up within a few days. Occasionally brownish discoloration may follow.

Herpetic lesions may be extensive enough to cause constitutional symptoms, malaise and

fever. Herpes virus has been reported as the cause of encephalitis.

Herpes simplex must be differentiated from herpes zoster, impetigo contagiosa, vesicular eczema, dermatitis venenata, and insect bites which cause vesiculation.

Many local medicaments have been used in the treatment of herpes simplex. The following are the most acceptable: spirits of camphor, ethyl alcohol (95 per cent), spirits of nitrous ether (full strength), aluminum acetate (2 per cent solution), and camphor phenol mixture. I have found the latter to be the most efficacious. The following formula is satisfactory for its preparation:

R ₁ Powdered camphor	3 drachms (12.00 gm)
Phenol crystals	2 drachms (8.00 gm)
Alcohol 95% to make	1 ounce (30.00 gm)
Mix	

Sig. Apply locally as needed.

Röntgen ray will relieve pain and itching but it is not to be used in recurrent cases. Codeine or other sedative may at times be necessary. Smallpox vaccination has been recommended in recurrent cases and in my experience has produced excellent results.

Geminate Ganglion Herpes (Hunt's Syndrome)—This is a type of herpes in which the pinna and external auditory canal are the site of a vesicular herpetic eruption, sometimes in association with herpetic lesions in the pharynx, in which there are also facial and auricular nerve palsies with associated pain, tinnitus aurium, and vertigo. It is a rare affection.

Herpes Zoster Oticus (Shingles)—Herpes zoster is an acute disease of the skin characterized by the occurrence of thick walled groups of vesicles which tend to remain discrete. It may affect the pinna and extend into the external auditory canal, or vice versa. The disease is probably due to a virus. Quite often it assumes the aspects of a mild epidemic. Pain, discomfort, hyperesthesia and formication are prominently associated with the disease and may even precede the occurrence of the vesicles. The vesicles appear in groups, have tough walls, may occur in successive crops, and are very prone to become secondarily infected. They recede by drying and usually some degree of scarring remains. The period of the acute phase of the disease varies from a week to ten days when the disease merges into a subacute phase in which the vesicles dry up and dis-

appear, leaving pitted and scarred hyperemic areas in the skin. Repeated attacks, while they do occur, are rare. The most distressing feature of the disease is the frequent sequela of pain and discomfort, burning or itching sensations in the previously affected areas. Such symptoms may persist for an inordinate length of time, sometimes many months, and prove very resistant to treatment.

In the acute stage of herpes zoster the treatment consists in the application of sedative and drying lotions. The orthodox calamine lotion, to which 0.5 per cent sodium sulfathiazole has been added, is a favorite. Ointments and greasy solutions are looked upon with disfavor by dermatologists in general. Local applications should not be allowed to accumulate on the skin, but should be gently cleansed away every day before fresh applications are made. Light application of the roentgen ray to the affected area of the ear is of some value in reducing pain. The use of narcotics should be guarded against, as they may be resorted to for relief of the distressing sensory phenomena of the convalescent stage which may last for months and lead to addiction. Surgical pituitrin, 0.5 to 1 cc hypodermically, has given good results in the hands of some observers. My experience with it in the treatment of herpes zoster has not been encouraging. Sodium iodide, 1 to 2 gm intravenously, once daily, is often of great value. Care should be observed to avoid administration of this drug to persons who are sensitive to iodine. Sensitivity can be determined by giving 10 to 15 grains of potassium iodide by mouth and observing the reaction for twenty-four hours before using intravenous iodides. Autohemotherapy is of value in most cases of herpes zoster. Daily doses of from 5 to 10 cc of the patient's own blood are injected intramuscularly. This may be alternated with the sodium iodide injections if desired.

Molluscum Contagiosum—The lesions of this disease may occur on the pinna. They consist of small cystlike structures which are ordinarily about the size of a pinhead at their outset but enlarge to varying degrees, sometimes attaining the size of a small pea. There is usually a central dimple or depression in the top of the lesions and they are filled with a semi-gelatinous fluid containing bodies designated as molluscum bodies. The disease is contagious and auto-inoculable. The lesions are usually multiple, and affect children and adults alike.

Diagnosis is established by the physical appearance of the lesions, which is characteristic, and is confirmed by finding the molluscum bodies in their contents.

The lesions respond readily to simple measures which consist of puncture or incision, expressing the contents, and application of some antiseptic, such as half strength tincture of iodine or trichloro acetic acid.

Chondrodermatitis Nodularis Chronica Helicis (Painful Nodule of the Ear)—Painful nodules occurring on the margin of the pinna without known cause may persist for years. Treatment is by cautery and curettage, or cautery alone.



Fig 219—Molluscum contagiosum of the pinna and adjacent scalp in a child. Other lesions were present on the neck and anterior wall of the chest. Another member of the family had a similar lesion on the lower eyelid of the left eye.

Atresia and Stenosis of the External Auditory Canal—Closure of the external auditory canal, either partial or complete, may be caused by encroachment of surrounding soft tissues or bone. The condition may be either congenital or acquired. The external meatus may be greatly reduced in size or completely closed. The entire external auditory canal may be congenitally absent and in such instances there is usually an associated absence of the deeper structures of the ear.

In congenital absence, where roentgenologic investigation and hearing tests indicate the probability of the presence of a functioning inner ear, an attempt at restoration of function may be made by forming a skin lined fistula.

back of the ear down to the mastoid antrum Attempts at the formation of a canal in its normal location are not recommended, as the results are wholly unsatisfactory The retroauricular fistulization operation is technically difficult and almost without exception the results are disappointing

Acquired atresia of the external auditory canal is usually the result of burns, injuries, or ulcerations about the ear If occurring at the entrance to the canal it may be overcome partially or wholly by excision of the encroaching tissue and the use of a skin graft of the Thiersch type on a stent of dental modeling compound of the desired size The skin graft is taken from the inner surface of the arm or thigh, and is applied, raw surface outermost, on the stent The graft thus prepared is introduced into the canal and allowed to remain in contact with the denuded area for a period of time sufficient for it to become attached, ordinarily from a week to ten days is required The result is usually an improvement over the original condition, and may for this reason be satisfactory, but the procedure may have to be repeated If the depths of the canal have been involved, the procedure outlined does not work well It is then best to amplify the size of the canal by removing the posterior bony wall in a manner similar to that used in performing the radical mastoid operation, incising the membranous portion of the canal and allowing the area to heal by granulation A Thiersch graft may likewise be employed in such instances, but is usually not necessary

Stenosis of the external auditory canal may result from thickening of soft tissues in chronic inflammations about the ear Long-standing inflammation of the skin of the external auditory canal will result in its thickening and subsequent narrowing A chronically discharging ear may produce such a reaction in the skin Various types of dermatitis of long standing may produce stenosis of the canal Lesions such as furuncles produce stenosis of the canal by the swelling associated with the acute process

There may be narrowing of the canal due to bony changes Fractures involving the walls of the canal, with subsequent bony overgrowth in healing, or the displacement of the fragments, may cause marked narrowing I have seen a single instance of unilateral hypertrophy of the mastoid process in an adult woman in which the canal was so narrowed by compression of its

posterior wall that only the smallest wire applicator could be introduced to cleanse it Bony overgrowths are sometimes annular, and this type is usually bilateral Exostoses are fairly common, and may seriously impair the lumen of the canal Ordinarily exostoses are asymptomatic, but in some instances may require removal

Treatment of constrictions or stenoses of the bony canal resolves itself in most instances into amplification of the bony canal by means of a modified radical mastoidectomy, removing as much of the posterior wall of the canal as is necessary The membranous canal may require a plastic procedure to adapt it to the enlarged bony canal, or a Thiersch graft may be required The membranous canal is easily stripped from the underlying bone, and when the bony tissue is removed it may be possible to cause the soft tissues to conform to the new bony canal through the simple treatment of packing In the annular type of stenosis, surgical interference will almost certainly damage the ear drum and a permanent perforation will remain This point should be thoroughly explained to the patient before any surgery is performed

Otalgia Due to Remote Lesions—Aside from the pain accompanying local lesions in the ear, referred pain is very important as its underlying factor may be serious and the pain in the ear will not be relieved unless the underlying cause is relieved

Pain referred from the throat is perhaps the most common of all types of referred pain in the ear The reflex is through the so-called Arnold's nerve, a branch of the vagus which supplies both the deeper structures in the throat and the skin of the external auditory canal Pain is an almost constant sequela of tonsillectomy, but it occurs also in acute tonsillitis, peritonsillar abscess, sphenopalatine neuritis, and nasopharyngitis It is a distressingly frequent attendant of laryngeal lesions, such as ulcers and new growths The so-called geniculate ganglion neuralgia is another type of referred pain Other frequent causes of reflex pain in the ear include dental caries, unerupted molars, traumatic occlusion of the teeth, and faulty closure of the jaws The latter condition may be due to abnormal wearing down of the teeth or misalignment such as would follow fracture of the jaw with faulty anatomical reposition of the fragments Overclosure of the jaws may be due to multiple extraction of teeth

It occurs frequently in completely edentulous persons for whom adequate and properly fitting dental plates have not been made. In overclosure, there is abnormal stress in the temporomandibular joint, which may become exquisitely sensitive to jaw motion. The immediate proximity of the ear to the temporomandibular joint is responsible for the frequent reference of pain to the ear in such cases. In the absence of acute processes in the ear, inspection of the mouth, combined with palpation of the temporomandibular joints, is usually sufficient to establish diagnosis. Measurement of the bite or closure of the jaw will establish the degree of deviation from normal. This is easily accomplished by use of the S. S. White Dental Bite Gauge. Roentgenograms of the joint may reveal bone changes in the advanced cases.

In referred pain to the ear, treatment obviously consists in care of the affection responsible. In pain referred from an inflamed tonsillar area, relief is obtained by applications of the usual time honored remedies such as 5 to 50 per cent silver nitrate solution, camphor-phenol mixture, 5 per cent mercuriochrome in 50 per cent ethyl alcohol, or phenol iodine and glycerin compound. The list of such remedies is quite lengthy, but the foregoing is sufficient to meet most requirements.

In seeking relief for otalgic pain following tonsillectomy, acetylsalicylic acid may be used. Silver nitrate applied to the fossa is also very satisfactory, but its effects are not immediate and quite often its use is undesirable because it temporarily intensifies the pain. As a local application, 1 gm of guaiacol in 1 ounce of olive oil is of value. Ethyl aminobenzoate produces a superficial but somewhat enduring local anesthesia when applied to the tonsil fossa. There are several proprietary preparations available in troche form in which ethyl aminobenzoate has been combined with other substances. These may be of service in relieving oral and pharyngeal reflex pain. Chewing gum containing acetylsalicylic acid is also available, but in the use of this, as well as the troches containing ethylaminobenzoate, caution is necessary to avoid gastric irritation which almost invariably follows the too liberal use of such preparations. Judiciously employed however, they have a definite place in our ther-

apeutic armamentarium. A combination local powder which has proven of value is

R: Menthol 1/10 gram (0.0065 gm)
Benzocaine t-2 grams (0.065 to 0.13 gm)
Cane sugar or milk sugar 10 grains (0.65 gm)
Mix, and place in no. 1 capsules
Sig: Dissolve on tongue or apply to area to be treated with powder blower

Codeine, morphine, or opium administered orally or in suppositories is always reliable for pain relief. The barbiturates are less profound in their action, but are particularly serviceable in suppository form when swallowing is difficult.

In the effort to relieve referred pain associated with laryngeal lesions such as tuberculous laryngitis and neoplasms, it is quite often necessary to block the superior laryngeal nerve with alcohol at the point where the nerve crosses the upper border of the thyroid cartilage.

In referred pain due to dental caries adequate dental attention is imperative.

The local use of heat is often of value, particularly in those cases due to lymphadenopathy in the immediate neighborhood of the ear. Moist heat is preferred for this purpose.

The use of warm olive oil in the ear may assist in relieving pain and is not objectionable under ordinary conditions.

WILLIAM DAVIS GILL

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EUSTACHIAN SALPINGITIS

Synonyms *Tubotympanic Catarrh, Catarrhal Otitis Media, Nonsuppurative Middle Ear Disease, Hydrops Ex Vacuo*

To understand the mechanics of inflammations of the eustachian tube and their influence upon the middle ear, both of these structures must be considered as integral parts of the same organ, anatomically, physiologically, and pathologically. The functions of the eustachian tube are (1) to equalize the air pressure in the middle ear and (2) to provide drainage. The lower two-thirds of the eustachian tube, from the isthmus at the junction of the osseous portion to the pharyngeal orifice (the *torus tubarius*), is a collapsed tube which normally opens only during swallowing and in conjunction with certain movements of the jaw as in yawning. The *schneiderian* membrane of the nose is continuous with that of the tube and middle ear which participate in inflammations of the nose and epipharynx. Beneath the mucosa of the membranous portion of the eustachian tube lie scattered masses of lymphoid tissue, usually more condensed in the lower part and particularly so in those individuals with a tendency to lymphoid hypertrophy, especially of the pharyngeal and faucial tonsils. These subepithelial lymphoid masses also participate in inflammations of the other portions of Waldeyer's ring.

The aerating function of the eustachian tube is to supply just sufficient intratympanic pressure to balance the external atmospheric pressure on the drum head, relatively 15 pounds to the square inch. In aviation and in carsson worker and other occupations, this atmospheric pressure varies greatly and, therefore, any factor which interferes with the ventilating function of the tube may spell disaster to the delicate middle ear mechanism.

Acute Phase—During an acute inflammatory process in the epipharynx or during an attack of allergic rhinitis, the mucosa of the pharyngeal orifice of the eustachian tube may swell partially or completely blocking this airway. If not speedily reopened, the oxygen content of the middle ear speedily becomes absorbed by the blood vessels in the mucosa of the middle ear with the creation of a partial vacuum. The symptoms of this phase are a fullness in the head, slight tinnitus, and slight impairment of hearing for the low notes. If the tube reopens

and air is admitted these symptoms immediately subside. If the tube remains closed for a considerable length of time there is a transudate from the blood vessels and glands of the middle ear mucosa. In the first or earliest stage, there may be retraction of the tympanic membrane, and in the later stages, if drainage continues to be obstructed, there may be bulging of the drum head with pain, even in the absence of infection. At any stage, infective agents, forced through the partially closed tube, or invading by the subepithelial route, as suggested by McMabon, may change the pathologic process into a suppurative otitis media. An attack in which supuration does not occur is often called "acute catarrhal otitis media." If the inflammation in the tubal mucosal or submucosal structures subsides sufficiently to restore aeration and drainage, the whole condition will subside.

Chronic Phase—The advance of the tubotympanic catarrh through the acute and subacute phases to the chronic phase depends upon (1) prolonged time of healing of the acute attack due to inadequate treatment or failure to eradicate the predisposing cause in the epipharynx, (2) repeated attacks, or (3) failure of complete resolution from the acute attack. Any of these result in the formation of fibrous tissue with consequent permanent narrowing of the eustachian tube (chronic adhesive process) and with the formation of adhesions within the middle ear, interfering with ossicular movement and with the vibrating quality of the membrane tympani and causing a permanent retraction of the drum membrane. This condition produces the so called "catarrhal deafness," also called "chronic catarrhal otitis media." This deafness is conductive in type, characterized, at least until the latter stages, by loss of bearing for the low tones, intact bone conduction and tinnitus.

Finally, in certain instances, the hypertrophic and obstructing condition just described is eventually succeeded by atrophy of the mucous membrane and underlying structures, resulting in a wide open, patulous tube, which transmits air ton freely. Deafness, autophonia, and aural discomfort with relaxation, abnormal motility, and atrophy of the drum head ensue. Fortunately this end result is comparatively rare.

Treatment—In the early stages, impaired hearing, due to chronic tubotympanic catarrh, as well as its accompanying tinnitus can often be relieved by appropriate treatment to the under-

lying causes in the nose and epipharynx and by careful treatment of the tube itself by inflation and dilation as outlined in other articles. In the late stages, it can be helped, at least temporarily, by insufflations of salicylic acid into the tube. Treatment may be given at monthly intervals, as recommended by Dr George E. Shambaugh, Jr.

Deafness in children, other than congenital, iuetic, or as a result of middle ear infection, is, according to Crowe and others, most often due to tubal catarrhal obstructions and is, surprisingly enough, characterized by the early loss of the very high tones, even to 16,000 double vibrations. In these cases, it is the lymphoid tissue in the lateral walls of the nasopharynx, as well as the submucosal lymphoid tissue in the tube itself, that is the etiologic factor. Since these small adenoid masses in and around the tube are beyond the reach of adequate surgery, their reduction can best be accomplished by the roentgen-ray, radium, or radon treatment. If properly carried out and before irreversible damage has taken place, restoration of hearing or prevention of further loss may be expected. This usually means before the twelfth year, thus early detection of impairment of hearing, especially in the upper registers, is imperative. This method of treatment is of little use in adults.

GEORGE MORRISON COATES
WILLIAM GORDON

TYMPANIC INFLATION

Tympanic inflation has a two-fold purpose (1) diagnostic and (2) therapeutic. Diagnostically, it helps to determine whether or not there is patency of the eustachian tube, fluid in the middle ear, and/or an invisible perforation in the drum membrane. Therapeutically it helps in maintaining patency of the eustachian tube, in stabilizing the air pressure in the tympanum, in freeing the tympanic cavity from secretions, and in the reduction of adhesions between the ossicles and tympanic walls.

Tympanic inflation of the middle ear is accomplished (1) by Valsalva's method, (2) by politzerization, and (3) by catheterization.

Valsalva's Method.—This procedure is limited in its usefulness, because it encourages an

act which has definite restrictions. One great advantage, however, lies in its simplicity; it may be performed by the patient. During the procedure the examiner can observe the mobility of the drum head through the external auditory canal. Small perforations, whose presence cannot be determined by inspection of the tympanic membrane alone, may be manifest and, if the middle ear is infected, fresh secretions may be collected for bacteriologic study, the preparation of a vaccine, or other purpose.

Under normal conditions, tympanic inflation can be easily accomplished by Valsalva's method, particularly if there is little or no congestion. However, if there is any swelling or congestion about the pharyngeal orifice of the eustachian tube, a greater force is naturally necessary for inflation. When too strong force is used, damage rather than good is done to the tympanic membrane. In persons with unilateral tubal disease, Valsalva's maneuver may produce a relaxation of the normal tympanic membrane because air will have been forced in through the good ear while attempting to ventilate the diseased ear.

The procedure is contraindicated in patients with high blood pressure, in those with infection in the nose (when active pus is present there is danger of infecting one or both ears), with cleft or frozen palate, with encephalitis, and in post-polio myelitis patients who have paralysis of the soft palate.

Technic.—By Valsalva's method, tympanic inflation is accomplished by the patient attempting to exhale forcibly while occluding both nostrils with the thumb and index finger and keeping the lips tightly closed. Air is thus condensed in the nasopharynx, it finds its way under pressure through the eustachian tube into the middle ear.

In the treatment of aero otitis media, Valsalva's method is modified by utilizing the air pressure machine. An olive shaped tip is attached to the pressure machine, inserted in the nostril, and the patient is instructed to close both sides of the alae nasi and continue swallowing for approximately twenty minutes, rest for one half hour, and then repeat this procedure until there is a feeling of the tube having opened. This method is substituted for that of interrupted valsolvization and politzerization.

Interpretation.—Subjectively, when the patient feels the air pressure in his ears one may conclude that one or both of the eustachian tubes are patent. In patent tubes one will notice the resilient movement of the drum head by its ballooning outward. If there is fixation of the tympanic membrane as in otosclerosis, or if there are adhesions of the tympanic membrane, there will be no movement of the tympanic membrane.

When air fails to enter into the closed or obstructed ear it may be assumed that the eustachian tube is closed, or that the concentrated air in the epipharynx is insufficient in volume to force the tube open. One must then resort to another method whereby inflation can be better accomplished

Politzerization—The physical principle of Politzer's method resembles that of Valsalva's method in that both procedures depend upon the condensation of air in the nasopharynx. Politzerization is accomplished by use of Politzer's bag or Politzer's inflating apparatus in conjunction with Valsalva's method.

Politzerization has a wider range for usefulness than does Valsalva's method. It is employed in patients with certain types of bilateral

tion. It may cause rupture of either the diseased or the normal drum head. In most instances it is the healthy drum head which is damaged. Where there is an acute upper respiratory infection or sinusitis there is danger of forcing the infected material through the eustachian tube into the middle ear. Politzerization is contraindicated in persons with high blood pressure, since the forcing of the air and the forced inspiration may create vertigo, dizziness, or an intracranial catastrophe. It is also contraindicated in patients with vertigo, in those with a cleft or frozen palate, encephalitis, or postpoliomyelitis associated with paralysis of the soft palate. It should not be utilized in the treatment of persons with true otosclerosis whose eustachian



Fig. 220—A, Valsalva's method of tympanic inflation. B, Politzerization.

ear disease, and in those with bilateral eustachian obstruction. It is effective in patients with nasal obstruction in whom the catheter cannot be utilized. It may be used in those with exudative and transudative catarrhal otitis media, and in the pre- and postoperative therapy of patients with mastoiditis to prevent adhesions and to remove the fibrinous exudate which is often present in the middle ear. Children will cooperate more readily with this type of technique than with Valsalva's method. Nervous patients also seem to respond more readily.

Politzerization is contraindicated in unilateral ear disease because the air is apt to be forced into the good ear, damaging a healthy tympanic membrane and creating undue relaxa-

tions. It cannot be employed in uncooperative patients.

Technic—The physician inserts the olive shaped nozzle of a Politzer's bag into one of the patient's nostrils and completely closes both the anterior nares with the thumb, index and third fingers. The patient is instructed to take a deep breath, close his mouth and puff out his cheeks. At the maximum point of the ballooning of the cheeks, the physician synchronously presses the bulb of the Politzer's bag thereby forcing air into the patient's epipharynx. If the technic is properly executed one or both of the patient's eustachian tubes will open and air will enter one or both middle ears. If air fails to enter it may be concluded that there is complete eustachian tube closure or that the procedure has not been properly conducted.

A variation of Politzer's method can be utilized. After inserting the nozzle of the Politzer's bag in the patient's nostril and closing the anterior nares, the patient is asked to swallow, and Politzer's bag is compressed when the patient's thyroid cartilage (Adam's apple) reaches the maximum point of elevation in his neck. The mechanical principle involved is that of the closing of the epipharynx by the soft palate, thus forcing the air through the eustachian tube. A further variation is the compression of the bag while the patient says "K-K-K-K" or the

gist the interpretation of the various sounds heard through the auscultation tube will aid in the diagnosis and the successful accomplishment of the therapy. Catheterization is of advantage in that the treatment can be limited to the ear involved, the air pressure can be regulated, and medication may be applied through the catheter. It is a selective qualitative and

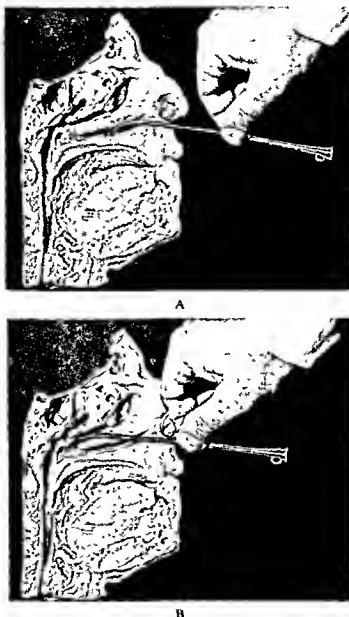


Fig. 221.—A, Insertion of catheter along the floor of the nose. B, Engagement of catheter in the eustachian orifice.

syllable "hawk." This maneuver also helps to close the epipharynx by the elevation of the soft palate, thus facilitating free passage of air through the middle ear.

Catheterization.—Catheterization offers the most satisfactory method of tympanic inflation for both examination and treatment in the greatest number of cases. To the experienced otolo-

gical method in determining the patency of the eustachian tube. By this method it can be determined whether the contents of the middle ear are dry or moist.

This type of inflation is contraindicated in the following conditions: deviated septa, spurs and ridges, vasomotor rhinitis, acute upper respi-

ratory infection, sinusitis, growths—benign or malignant—nasal polyps, polypoid degeneration of the posterior ends of the inferior turbinates, hypertrophied adenoids, and where the nares are too narrow to insert a catheter. It is also contraindicated in infants and children and should not be employed in nervous patients. Through catheterization infectious material may be blown into the middle ear. Trauma to the mouth of the eustachian tube may be inflicted, creating edema and synechia. Emphysema of the soft tissues and the neck may result from improper technic. Catheterization may cause pain.

Technic—Local anesthesia is employed. Cocaine (5 per cent) in adrenalin (1:20,000) may be used. However, since the treatment may be given frequently, I prefer pontocaine (2 per cent) in neosynephrin (1 per cent), thus avoiding the possibility of the patient becoming addicted to cocaine. Under direct vision and with proper lighting an applicator with a cotton pledget, which has been dipped in the anesthetizing solution, is placed along the floor of the nose and allowed to remain there for about five minutes. A curved applicator, which has also been dipped in the solution, is then gently inserted into the floor of the nose until it reaches the epipharynx. This applicator is turned with the beak outward in the direction of the lateral nasal wall. It is then pulled forward until it is engaged in the mouth of the eustachian tube. Thus the area is properly anesthetized, the orifice of the eustachian tube is decongested, and gagging which often is so annoying to the operator as well as to the patient is avoided. The patient is also relieved of pain and the possibility of bleeding is greatly lessened.

A No. 2 silver malleable catheter is usually employed. It is heated slightly, greased, and the curve is molded to conform to the individual patient. Under direct vision, with proper lighting and with the aid of a nasal speculum, the catheter is inserted carefully and gently along the floor of the nose until it reaches the posterior pharyngeal wall. It is then turned with the guide ring outward, thus indicating that it is in a lateral position. The operator then pulls the catheter slightly forward until the beak is engaged in the fossa of Rosenmüller. Then, with another tug, the catheter is again pulled slightly forward. When one feels that the beak is overriding the posterior lip of the eustachian tube and is engaged in a depression the otologist knows that the catheter is in apposition with the orifice of the eustachian tube and catheterization can then be attempted. One end of an auscultation tube is placed in the patient's ear and the other in the operator's ear. Compressed air may be forced through the catheter either from a Politzer's bag or from air pressure equipment, or whatever pressure equipment is on hand.

When treatment of the eustachian tube is indicated and there is an obstruction of the nose on the same side, one may catheterize through the opposite or unobstructed side of the nose. In this case the catheter is placed along the floor of the nose until it reaches the posterior wall of the pharynx. The catheter and the guide ring are then rotated to the diseased side. The

beak of the catheter is then in the choana posterior to the septum. The guide ring is then pointed in the direction of the outer canthus of the eye of the diseased side. When the catheter is pulled forward it first engages in the fossa of Rosenmüller and when pulled forward again it is in apposition with the orifice of the eustachian tube. Inflation and auscultation may then be carried out as previously described.

In the event that one cannot locate the orifice of the eustachian tube blindly one can utilize the nasopharyngoscope by placing it in the opposite side of the nose and observing whether or not the catheter is properly engaged.

Interpretation—When the eustachian tube is patulous the examiner can hear, transmitted through the auscultation tube, a low pitched tone resembling the sound of "who" When there is a partially obstructed eustachian tube he will hear a high pitched sound. When there is a completely closed tube no sound will be heard at all, with the exception of that due to vibrations of the tissues, this will also be true when the catheter is not engaged in the orifice of the eustachian tube. In the presence of fluid one may hear a gurgling or a rumbling sound. When there is a small perforation a high pitched whistle will be heard.

In the event that ventilation in the middle ear cannot be accomplished by any of the three methods enumerated (Valsalva, politization and catheterization) one must conclude that the eustachian tube is completely closed either because of hypertrophy, strictures, synechia, or other condition, in such event eustachian bougienage is indicated.

MATTHEW S. ERSNER

EUSTACHIAN BOUGIENAGE

The normal eustachian tube is patent, but the musculomembranocartilaginous portion of the tube remains closed by the apposition of its walls and opens only upon yawning, sneezing or swallowing, and when air is replaced in the middle ear. Such actions are partly voluntary in nature. During disease, however, the eustachian tube may be closed, as the result of active or passive congestion, or because of hypertrophy of the mucous membrane, mucosa, and submucosa. Closure of the eustachian tube may also be due to synechia, adhesive bands, strictures, or stenosis at the isthmus. Thus, when the tube is not patent, from whatever cause, measures must be instituted for correction of the condition. For example, the mucous membrane is

decongested by the application of medicaments the tube is stretched and dilated by insufflation or adhesions and strictures may have to be broken up in order to re establish the

to ventilate the middle ear by Valsalva's Politzer's or the catheter method (3) hypertrophy of mucous membrane (4) active or passive congestion in the eustachian tube (5) adhesions



Fig 222.—The Yankauer bougie inserted into a no 2 catheter and engaged in the mouth of the eustachian tube

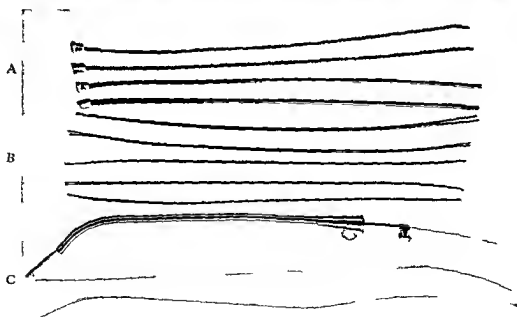


Fig 223 —A Soft rubber sounds B whalebone filiform bougies C Yankauer wire bougies.

lumen to permit aeration and ventilation of the middle ear

Eustachian bougienage is indicated when one or more of the following conditions exist (1) obstruction of the tube (2) where it is impossible

synechia and strictures (6) impairment in hearing (7) progressive hearing loss (8) tinnitus (9) vertigo due to the disturbed intratympanic pressure secondary to tubal closure It is contra indicated (1) when the tonsils and adenoids are

hypertrophied, (2) in acute upper respiratory infection, (3) when there is free pus, (4) when acute rhinitis is present, (5) when there is virus infection, (6) in the exanthemas, (7) in allergy, (8) when sinusitis exists (9) when there are benign or malignant growths in epipharynx, (10) when polyps are present, (11) when there is polypoid degeneration of the posterior ends of the inferior turbinates and (12) in septal deformities, such as spurs, ridges, and deflections involving the cartilaginous and osseous portions.

Carrying infection into the eustachian tube and middle ear should be avoided as should traumatization of the tubal tissue, which might lead to infection, synechia and stenosis. There

The following are the various types of bougies used: (1) the Yankauer wire bougie, (2) soft rubber sounds, (3) the whalebone filiform bougie (Urbanschitsch's eustachian bougie) (See Fig. 223).

Yankauer Bougie.—In 1910 Yankauer suggested the use of twisted wire applicators with a loop at one end to hold the cotton and medication as bougies. This applicator is held in place by a handle which is marked in centimeters. A no. 2 eustachian catheter is employed after it has been sterilized, dried, cleaned, and freed of all debris. The Yankauer bougie in its special handle is inserted into the catheter. The catheter is inserted along the floor of the nose until it reaches the posterior pharyngeal wall; at this point it is rotated so that the guide ring points outward. The bougie is gradually worked through the catheter until it passes through the congested area, stricture or isthmus in the eustachian tube. The nasopharyngoscope may be em-

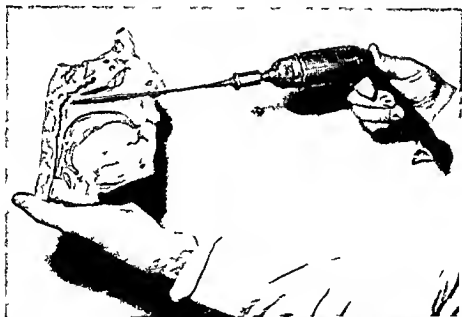


Fig. 224.—The Coates special heating apparatus for insufflation of medicated vapors.

is also danger of emphysema of the neck and soft tissues if the bougie should accidentally puncture the soft tissues about the eustachian tube. (Note: Inflation should not be attempted if there is any suspicion that the soft tissues have been penetrated.) A remote danger is that of injury of the carotid artery which lies medially to the eustachian tube. The applicators should be tested before they are inserted, thus minimizing the danger of breakage while they are in the eustachian tube.

Technic.—The nose is typically anesthetized along the floor and along the path of the catheter. With the aid of a curved applicator the orifice of the tube is also anesthetized. Cocaine (5 per cent), cocaine (2 to 5 per cent) in 1 per cent ephedrine, pontocaine (2 per cent) in 1 per cent neosynephrin, or hulesin may be employed.

employed as a guide. The bougie is allowed to remain in the tube for one half hour, then both bougie and catheter are removed. After the tube has been dilated one may catheterize it and insufflate it with air pressure. After dilatation various medicaments or vapors may be beneficially applied or insufflated through the catheter. Inflation must not be attempted if there is any suspicion of injury due to faulty technique, since emphysema may result. (See Figs. 222 and 223C.)

Soft Rubber Sounds.—The soft rubber sounds (Fig. 223A) are of different thicknesses and have various markings. The appropriate sound is placed into the eustachian catheter and gradually inserted and passed through the mouth of the eustachian tube until it has gone beyond the point of obstruction. It must be constantly borne in mind that the eustachian tube is only from 1 to 1½ inches in length and that the sound must not be inserted and passed in for more than one inch.

Whalebone Catheter.—The technic of eustachian bougienage with the whalebone catheter (Fig. 223B) is the same as that with the soft rubber sound.

Drugs Employed—Peritubal and/or intratubal medication may be applied

For *peritubal medication* one of the following is used (1) boroglycerate of tannin (5 to 10 per cent), (2) a weak solution of iodine and potassium iodide (2 to 5 per cent), (3) a 1 per cent solution of silver nitrate

For *intratubal medication* shrinking solutions such as 1 per cent ephedrine sulfate, 2 per cent cocaine in epinephrine hydrochloride (1:30,000), and 1 per cent neosynephrin may be used. Benzodrine may be insufflated through the catheter. Helium gas inflation may be effective. Heated medicated air may be blown through the catheter. Dr. George M. Coates has devised a special apparatus for the insufflation of medicated vapors (see Fig. 224)

A small amount of the following powder is put between small layers of cotton and placed in the Coates apparatus where it is electrically heated and thereby converted into vapor

R	Iodine crystals	5 grains (0.3 gm)
	Menthol crystals	5 grains (0.3 gm)
	Sodium bicarbonate	$\frac{1}{2}$ ounce (14.3 gm)
	Talc	$\frac{1}{2}$ ounce (14.3 gm)

By means of air pressure the vapor is insufflated through the catheter into the eustachian tube and middle ear

MATTHEW S. ERSNER

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NONSUPPURATIVE DISEASES OF THE MIDDLE EAR

There are two types of these so-called "catarrhal" diseases, the *secretory transudative noninflammatory* type and the *secretory exudative inflammatory* type. Although both the transudative and the exudative types begin as nonsuppurative affections of the middle ear and the eustachian tube and may terminate as such,

some otologists prefer to identify these types as (1) the dry, and (2) the moist. Both the noninflammatory and the inflammatory types have in common the collection of fluid in the middle ear, with a closure of the eustachian tube. This condition may be transudative and noninflammatory, or may subsequently become exudative and inflammatory

SECRETORY TRANSUDATIVE NONINFLAMMATORY MIDDLE EAR DISEASE

(*Synonyms* *Hydrotympaum*, *Transudative "Otitis Media," Secretory Catarrh, Serous Catarrhal "Otitis Media"*)

Secretory transudative noninflammatory middle ear disease is characterized by a collection of fluid in the middle ear which is clear, brothlike in nature, and does not contain any of the elements of inflammation

Etiology.—The predisposing causes of this disease are varied. The condition is most common in the aged, but is often found in infants. It is also common in the youth of today who are in the aviation forces. Climate and change in humidity have a bearing on its development as does occupation. Asthenia and the dyscrasias, such as leukemia, render an individual especially susceptible. The condition may develop from continuity through the nasal passages, the middle ear becoming affected, or the eustachian tube and the middle ear may in themselves have become affected. Irritation of the eustachian tube and the stimulation of the secretory glands in the membranocartilaginous portion of the eustachian tube with closure of the pharyngeal orifice of the tube may be responsible. The affection may be secondary to a mild rhinitis, low-grade sinusitis or a low-grade rhinopharyngitis. It may be attributed to peritubal adenoid tissue, allergic or metabolic disturbances, gout, or arthritis. It may follow idiopathic closure of the eustachian tube, passive congestion resulting in osmotic hypovirulent infection, or "aero-otitis media."

In *aero-otitis media* there is often found a collection of serosanguineous fluid in the middle ear which is due to rapid descent of the plane causing an increase in the atmospheric pressure, with a resultant intense closure of the eustachian tube. A subsequent vacuum is thereby created, resulting in hypersecretion, osmosis, capillary rupture, and occasional frank bleeding in the middle ear.

Pathology—One or more of the following may be the pathologic factor or factors in the development of secretory transudative noninflammatory middle ear disease (1) closure of the eustachian tube, (2) vacuum (3) hypersecretion of the glands in the membranocartilaginous portion of the eustachian tube, (4) venous stasis of the eustachian tube and the middle ear, (5) osmosis, due to stasis and vacuum, (6) a collection of a sterile, clear, brothlike fluid with a low albumin content

nose is often heard. Should the eustachian tube become patent, the fluid will empty itself and the patient will complain of hearing a sharp high pitched whistle.

Examination and Diagnosis—The tympanic membrane is pearly gray in color, or occasionally, lusterless or ashen. All the normal landmarks are present. During the early stage of the disease there may be a retraction of the tympanic membrane, due to the closure of the eustachian tube. When there is a scanty amount

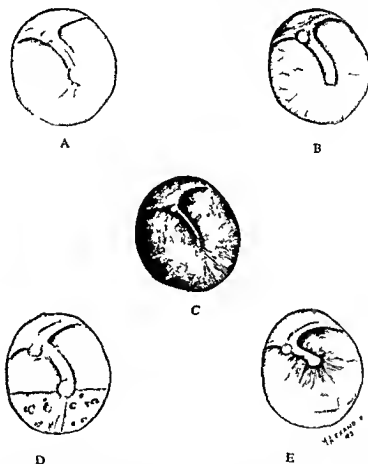


Fig. 225—A Normal tympanic membrane B Acute myringitis C Bulging drum head with inflammatory exudate D Secretory noninflammatory Calcareous transudation E, Retracted tympanic membrane with cal careous infiltration and adhesive processes in the middle ear

Symptoms—The symptoms vary according to the period of time which has elapsed since the onset of the disease. During the acute stage there may be pain and moderate fever. One or both ears may become blocked. The hearing may be impaired. There may be autophonia. At times there may be dysacusia. Mild vertigo is sometimes noted. There may be a sensation of water in the ear. A gurgling sound which becomes aggravated upon forcible blowing of the

of fluid in the hypotympanum no changes may be noted upon otoscopy. When the fluid increases a fluid level or a meniscus may be noted. A hairline fluid level will be noted depending upon the quantity of fluid and air present in the middle ear. Movements of the patient's head will reveal fluid level changes on otoscopy. When the fluid fills the middle ear completely, various degrees of fulness or bulging of the tympanic membrane will be noted. Bubbles

may be noted through the tympanic membrane in the course of Valsalva's procedure, as air enters the middle ear through the eustachian tube. On auscultation, a gurgling, rumbling sound is heard when catheterization is successfully accomplished. Hearing tests will reveal the presence of a mixed type of deafness, conductive and perceptive. Audiometrically there is a loss of hearing ranging between 20 and 30 decibels, the low and high frequencies are involved. Tuning fork and speech tests will substantiate the audiometric findings.

Treatment—The earlier the treatment is instituted the more likelihood is there of a favorable outcome. Several factors should be considered in the treatment during the acute stage. Primarily, the cause should be sought and, if possible, removed. Supportive treatment is instituted through the medium of hemotonics, tonics, vitamins, diet, alteratives, salicylates, and/or sulfonamides. Atropine sulfate in physiologic doses is a most potent and useful drug. It helps to dry up all secretions and is especially valuable when there is hyperactivity of the gland around the eustachian tube. Rhinologic treatment should include treatment of the sinuses and the use of decongestant solutions, such as cocaine (2 to 5 per cent), ephedrine sulfate ($\frac{1}{2}$ per cent) in normal saline solution, neosynephrin ($\frac{1}{2}$ to 1 per cent), or pontocaine (2 per cent) in neosynephrin (1 per cent) when anesthesia and a decongestant are essential. Decongestant solutions in combination with the sulfa drugs may be employed. Proetz displacement treatment may be utilized. The mouth of the eustachian tube should be shrunken, this may be accomplished with or without the aid of a local anesthetic. Tympanic inflation by Valsalva's method, politization, or catheterization should usually be attempted, when atmospheric pressure fails. Inhalation with helium may be successful. Physiotherapy will help absorption in chronic cases, ultraviolet, infra red, or diathermy treatment, or x ray exposure of the epipharynx and eustachian tube may be utilized. Radium may be applied directly against the mouth of the eustachian tube especially when there is evidence of adenoid tissue, excessive glands, or hyperactivity of the mucous glands.

Myringotomy is indicated if other measures have failed. The following technic has been found successful:

A semicircular incision is made, beginning in the region of the superior posterior portion of the tympanic membrane and extending downward toward the inferior quadrant. Aspiration and suction through a cannula should follow immediately.

Pneumomassage is imperative and should always be instituted to prevent adhesions and impairment in hearing. No aural medications, such as drops or irrigation, should be used since they might lead to infection.

Sequelae.—Resolution usually takes place if treatment is instituted early. If there has been any lapse of time before treatment is commenced, certain changes may have taken place. When there has been inflammation, secretory exudative inflammatory otitis media may result. Acute suppurative otitis media likewise may be a sequela. Impairment in hearing, varying from mild to profound, may take place. There may be transitory aural vertigo or recurrent pseudo-Meniere's syndrome. Atrophic and hypertrophic changes of the tympanic membrane may become evident.

SECRETORY EXUDATIVE OTITIS MEDIA

(Synonyms *Tubotympanitis*, *Catarrhal Otitis Media*, *Subacute Catarrhal Otitis Media*, *Exudative Otitis Media*)

Secretory exudative inflammatory otitis media is characterized by a collection of fluid in the middle ear which is turbid and which contains elements of inflammation.

Pathology—This type of otitis media may be serofibrinous or exudative. It may be defined as a vascular exudate possessing degenerative and regenerative phenomena. This occurs in living tissue as a result of irritation, which may be infectious, mechanical, thermal, or chemical, and may be transmitted by continuity or contiguity. The evolutionary pathologic mechanisms are (1) temporary contraction of blood vessels, (2) dilatation of blood vessels, venules, and arterioles, (3) vascular stasis, (4) lymphatic circulatory interruption, (5) round-cell infiltration consisting of polymorphonuclear leukocytes and a small number of lymphocytes, (6) histocytic infiltration, (7) exudate consisting of albumin and fibrin, (8) attempt at connective tissue proliferation. Or the pathologic process may be summarized thus: congestion (active and passive), exudation, cellular infiltration, formation of new capillaries, formation of fibroblasts with attempted multiplication of fibroconnective tissue cells.

Etiology.—In a large measure, the same etiologic factors are responsible for the exudative inflammatory type of middle ear disease as are accountable for the transudative noninflammatory type. These include acute upper respiratory infection, virus infection, acute rhinitis, recurrent rhinitis, influenza, acute and chronic sinusitis (isolated or pansinusitis), suppurative or nonsuppurative sinusitis (hypertrophic or hyperplastic), rhinopharyngitis, hypertrophied tonsils and adenoids, the exanthemas, tuberculosis, allergy (perennial and seasonal), swimming, improper blowing of nose, nasal irrigations, pathologic nasal conditions (such as septal deformities, polyps, hypertrophied turbinates, polypoid degeneration of the posterior end of the inferior turbinate, or benign or malignant growths), improper practice of middle ear ventilation (such as faulty technic in inflation by Valsalva's method, catheterization, or politizerization).

Symptoms.—The chief subjective symptoms are impaired hearing and tinnitus. The impairment in hearing may be slight or marked. There is usually a feeling of stuffiness or blocking in one or both ears. Some patients complain of slight pain. There may be autophony or dysacusia. Mild or severe vertigo may be experienced. Upon blowing the nose, the patient often feels a sensation of relief as the result of air having entered the middle ear, or pain may become aggravated, due to the forcing of infected material into the middle ear, or because of an increase in the intratympanic pressure caused by the blowing of the nose. There may be lassitude, slight or low-grade temperature, malaise, headache, and indisposition or general symptoms may be completely absent.

Examination and Diagnosis.—Anterior and posterior rhinoscopy may or may not reveal the presence of any abnormality. Nasopharyngoscopic examination may or may not disclose congestion of the epipharynx. There may be a swelling and congestion about the eustachian tube with closure of the tube. One may note pus, hypertrophy, growths, polyps, polypoid degeneration of the posterior ends of the inferior turbinates, lymphoid tissue, or other abnormal conditions.

On otoscopic examination the tympanic membrane appears diffusely red during the early stage of the disease, and it may also be retracted (see Fig. 225, B and C). Normal landmarks are absent. Later the drum head may be

edematous or it may actually bulge. Use of the Siegle's otoscope and pneumomassage will demonstrate whether or not it has mobility and elasticity.

The eustachian tube may be patent, partially patent, or completely closed, as revealed upon tympanic inflation by means of Valsalva's method, politizerization, or catheterization with auscultation.

Treatment.—The treatment should be based upon consideration of the etiologic as well as the pathologic changes that have taken place. Constitutional treatment consists in rest in bed when possible, treatment of the underlying disease, the administration of a cathartic, the institution of a proper diet, the promotion of diaphoresis, alkalinization (where acidosis is suspected), the administration of iodides to help absorption, and steam inhalation. The sinuses are treated, decongestant solutions, such as cocaine (2 to 5 per cent), ephedrine sulfate ($\frac{1}{2}$ per cent) in normal saline solution, neosynephrin ($\frac{1}{2}$ to 1 per cent), or pontocaine (2 per cent) in neosynephrin (1 per cent), should be used. The eustachian orifice and tube should be treated. Shrinking solutions are applied in the region of the eustachian orifice. The solutions may be combined with local anesthetics, such as cocaine (4 per cent) or pontocaine (2 per cent). Glycerate of tannin (5 to 10 per cent), zinc chloride solution (1 to 2 per cent), silver nitrate ($\frac{1}{2}$ per cent), or epinephrine hydrochloride (1:20,000) may be applied directly into the tube.

Treatment of the ear itself is accomplished through the following means:

1 Copious irrigations are given with a fountain syringe. One may employ a hot saline, a hot boric acid, a hot phenol (1:600), or a cresalin compound (1 teaspoonful to 1 quart of water) solution.

2 If there is pain, drops may be used. Those composed of a mixture of glycerin and phenol (5 per cent) are best. Auralgan may be somewhat disadvantageous in that it has the tendency to produce blebs or bullae on the external portion of the tympanic membrane in certain cases.

3 Chewing gum and swallowing the saliva should be encouraged after nasal and tubal decongestion, in the hope that this will help promote patency of the eustachian tube through the movement of the soft palate and the traction of the various muscles of the tube.

4 A hot gargle may afford relief

Before attempting tympanic inflation, one must make certain that the nose and paranasal sinuses have been completely cleared and are free of pus. This may be accomplished by shrinkage, irrigation, and suction. Tympanic inflation is accomplished by Valsalva's method, Politzerization, or catheterization, or eustachian bougienage is utilized when the other methods of inflation have failed.

Pneumomassage and diathermy treatment may be of benefit. Myringotomy is performed when necessary to permit the escape of the exudate and prevent formation of granulation tissue, adhesions, adhesive bands, and permanent retraction of the tympanic membrane, with resultant deafness. It should be performed under strictly aseptic conditions and may be accomplished under either local or general anesthesia.

Prognosis—Prognosis depends entirely upon the duration of the chronicity and the pathologic changes that have taken place in the tympanic cavity. If the disease is of long standing, accompanied by pronounced degenerative changes, the prognosis cannot be too favorable. However, if the condition has not been neglected the possibility of the restoration of hearing is favorable.

Termination—There may be complete resolution. The most prevalent complication may be chronic adhesive processes in the middle ear, particularly when there is recurrent exudative ("catarrhal") otitis media. There may be impairment of hearing varying from slight to marked deafness. The drum membrane may perforate spontaneously. Acute or chronic suppurative otitis media may be a sequela as well as mastoiditis with or without attending complications.

CHRONIC ADHESIVE PROCESSES IN THE MIDDLE EAR

(Synonyms: Chronic Middle Ear "Catarrh," Otitis Media Catarrhalis Sicca, "Dry Catarrh" of the Middle Ear, Proliferative Inflammation of the Middle Ear)

In any discussion of chronic adhesive processes in the middle ear it must be understood that this type of condition is a gradual development in the course of chronic "catarrh." It is difficult to determine exactly where exudative inflammatory disease of the middle ear ends

and adhesive processes of the middle ear begin. We therefore assume that there is a gradual conversion from an exudative inflammatory state to a hyperplastic condition in which there is a different pathologic process requiring a different type of treatment.

Etiology—Very often the lesion is so advanced when first seen by the otologist that it is next to impossible to determine the original cause. The condition is most frequently found in adults. It may be a sequela to continued or repeated attacks of subacute exudative or moist "catarrh" of the middle ear or eustachian tube in which resolution has never been quite complete, or it may follow recurrent attacks of acute rhinitis, each attack having produced greater tubotympanic congestion. Nasopharyngeal lesions obstructing the eustachian tube, with further involvement of the tympanum, may be responsible. Other predisposing factors may be those listed under "Secretory Exudative Inflammatory Otitis Media."

Pathology—The underlying pathologic factors are those of chronic inflammation with structural change in the mucous membrane. There is an increase in the substance caused by round cell infiltration with mature fibrous connective tissue. In some cases the thickening not only affects the lining of the membrane of the tympanic cavity but also extends to the ossicles and covering of the capsular ligaments. There are localized extensive adhesions between the membrana tympani and the inner wall of the tympanic cavity. At times there is a complete obliteration of the attic. The power of vibration of the sound-conducting apparatus is more or less impaired because of the changes in the tympanic cavity. There is ankylosis of the ossicles. Pathologic changes are manifest around and in the fenestra (oval and round window). Changes are present in either or both eustachian tubes decreasing the power of ventilation of the middle ear.

Symptoms—Subjective symptoms consist in impairment in hearing (usually conductive in nature but may be perceptive or a combination of conductive and perceptive), tinnitus, transient vertigo, and lightheadedness or giddiness. There is absence of pain.

Examination and Diagnosis—Upon otoscopic examination the drum head appears featureless (see Fig. 225E). Its landmarks are distorted. Retractions vary and are dependent upon the location of adhesions. The manubrium

um mallei may be prominent There may be prominence of the short process of the malleus The umbo may be displaced There may be dis

may be retraction of the tympanic membrane on each side of the malleus Shrapnell's membrane may be distorted and retracted

DIFFERENTIAL DIAGNOSIS

	Secretory Transudative Noninflammatory Middle Ear Disease	Secretory Exudative Otitis Media	Acute Suppurative Otitis Media	Otitis Tympano-bullosa	Hemotympanum
Color of drum bead	Pearly gray	Reddish	Red or gray, depending upon contents in the middle ear	Bluish	Dark red
Inflammation	None	Mild	Present	Present	None
Congestion	None	Mild	Marked	Marked	None
Swelling	None	Mild	Marked	Marked	None
Landmarks	Present	Distorted	Absent	Distorted	Absent
Bulging	None	May or may not be present	Present	Bullae are bulging but not tympanic membrane	May or may not be present
Fluid level	Present and visible	Not visible	Not visible	Not visible	Not visible
Air bubbles	Present	Absent	Absent	Absent	Absent
Pus	Absent	Absent	Present	Serosanguineous	Absent
Response to atropine sulfate	Present	Absent	Absent	Absent	Absent
Myringotomy	Escape of clear fluid	Turbid fluid released	Pus released	No myringotomy but puncture of bleb discloses escape of serosanguineous fluid	Bleeding or in spissated blood
Temperature	Normal	Low or normal	Elevated	Elevated	Normal
White cell count	Normal	Low or normal	Leukocytosis	Leukopenia when influenzal, otherwise a leukocytosis	Normal
Organisms	None	None	Present	None or influenzal	None

(From Ersner Matthew S. Diagnosis of Otic Surgical Lesions in Nelson's Loose Leaf Surgery of the Ear, New York, Thomas Nelson & Sons 1938)

placement of the cone of light either forward or backward, or the cone of light may be absent. Prominent malleolar folds may be noted. There

Examination of the mouth of the eustachian tube by means of the nasopharyngoscope or mirror may show indications of chronic congest-

tion, thickening, improper opening and closing of the tube, synechia, scars, and/or adhesions. There may be difficulty or inability to ventilate the middle ear by Valsalva's method, politzerization, or catheterization. Probing may disclose narrowing of the eustachian tube, and it may be difficult to pass sounds or bougies through the tube. There may be thickening of the mucosa or submucosa or actual strictures resulting from synechia and adhesions.

In *conductive deafness* there is diminished air conduction and loss of perception for low tones. Bone conduction, as determined by Schwabach's test, is good. In Weber's test there is lateralization of sound to the affected side. During the early stage of the lesion reaction to Rinne's test is positive, and the reaction diminishes as the disease progresses. Audiometric findings will disclose loss in the low frequencies but hearing within the speech range may not be affected.

In *perceptive deafness* there is a loss in the high frequencies. Schwabach's test shows diminished values. In Weber's test there is lateralization of sound to the good ear. Reaction to Rinne's test is negative. Audiometric findings will disclose loss of hearing in the high frequencies beginning with 4096 and upward.

In *mixed deafness* (conductive and perceptive), there is profound impairment in hearing. Both air and bone conduction are diminished. There is loss of hearing in both the low and high frequencies. These findings can be corroborated by audiometer, speech, whisper and spoken voice, and tuning fork tests.

Treatment.—Nonsurgical treatment consists in inflation of the middle ear by Valsalva's method, politzerization, or catheterization, treatment of the eustachian tube, pneumomassage, roentgen-ray therapy, bougenage. Hearing aids (air or bone conduction) may be recommended.

The operative treatment for chronic adhesive processes in the middle ear and its attenuating results is not very encouraging. The problem confronting the otologist is to keep the tympanic window open and to prevent new adhesions from forming. For many years Dr. George M. Coates and I have treated the condition by making an incision in the drum head and creating a window through the tympanic membrane. We have cut the adhesions so as to relieve traction upon the tympanic membrane. In a few instances, improved hearing has resulted,

but such relief has usually been only temporary and transitory in nature. The tinnitus also has been occasionally relieved but this too has been temporary. We have now abandoned the surgical treatment because of the fact that there is rapid closure of the wound and new adhesions form which are sometimes worse than the old. Also there has been the occasional complication, following this operation, of a suppurative otitis media. We have tried Thiersch's skin graft, silver nitrate application, and roentgen-ray therapy in an effort to keep the tympanic fenestra open. Unfortunately, we have been unable to accomplish this permanently and improvement in hearing has failed. Therefore, we do not recommend surgery on the tympanic membrane in adhesive processes in the middle ear unless there is an element of otosclerosis, in which case labyrinthine fenestration may be considered.

Prognosis.—The prognosis is favorable when there is response to inflation of the middle ear and impairment in hearing is not marked. It is unfavorable when there is pronounced deafness.

MATTHEW S. ERSNER

THE TREATMENT AND PREVENTION OF DEAFNESS

General Considerations — Causes of Deafness

—Years ago, Emerson taught that a primary focus of infection exists for most cases of deafness in which the perceptive element predominates. He believed that this primary focus might be subject to repeated acute exacerbations or remain quiescent, running a chronic course, but elaborating toxins that were slowly absorbed, producing auditory damage. In other words, the entire picture was to be looked upon as one of toxemia, septicemia, or pyemia, following an acute or chronic pattern. The focal processes might often be multiple and might be found in the teeth, tonsils, sinuses, and in the lymphoid elements of the epipharynx, oropharynx, and laryngopharynx as well as in and around the lateral pharyngeal folds and Rosenmüller's fossae. In searching for foci, the intestinal tract, the appendix, the gallbladder, and the colon should not be overlooked. Later,

Emerson stated that, essentially, the perceptive apparatus is the mechanism involved in the chronic type of deafness in which there is a tendency toward further steady or intermittent loss of hearing, of variable cause, in persons with or without a history of previous suppuration, and that the focus might be even a closed follicle in a chronically infected tonsil. He further taught that pathologic processes in the tube and middle ear had little relation to nerve dysfunction. However, he advised the removal of every vestige of infection particularly emphasizing the infective processes in the nasopharynx, lymphoid tissue, and bands between the eustachian tube orifice and the pharynx, and degenerative tissue in Rosenmüller's fossae, as well as thorough removal of the faucial tonsils.

Tilley has stated that when defective sinus drainage exists, free, spontaneous, permanent drainage should be instituted at once. The nasal accessory sinuses should be studied clinically as well as by roentgen ray, and the latter study augmented by use of radiopaque oil injected directly into the maxillary sinuses, and instilled in the posterior ethmoids and sphenoids by the displacement method of Proetz. By these measures a latent sinusitis may be discovered, the treatment of which might well prevent a certain amount of deafness.

Emerson has again emphasized the importance of the maxillary sinus as a focus of infection, which coincides with our observations.

Mosher feels that although we can by routine examination estimate the gross amount of infection in a given antrum, we can by no definite means evaluate the ability of any given mucous membrane to produce toxins. Furthermore, it is not the amount of toxins, but their virulence, which determines whether the effect caused by them is local or general. Mosher does not believe that pus and polyps in the antrum cause very much trouble, but that they are, as a rule, self-limited conditions. Lastly, he states that when a mucous membrane has reached the atrophic stage it usually becomes innocuous, incapable thereafter of acting as a focus of infection. Emerson, however, takes the stand that even an atrophic mucous membrane, as well as its periosteum, may act as a focus of infection.

Sturm has been impressed with the fact that chronic progressive deafness begins in childhood. He stated that he had rarely seen a perfectly normal drum membrane in a child with adenoid masses. He divided adenoid deafness

into three stages: occlusion, transudation, and cicatrization. In a study of 200 patients suffering from deafness, he found 32 per cent owing their incurable disability to nasopharyngeal disease in childhood.

Crowe is of the opinion that many cases of adult deafness have their genesis in childhood. He has conducted for a period of some sixteen years, for research purposes, complete studies on more than 15,000 patients including audiometer, tuning fork, and voice tests. Approximately 3000 of these patients were children. These tests were made in a soundproof room with 1 A Western Electric audiometer with a range of from 32 to 16,384 double vibrations. A high tone deafness was found. From these observations Crowe tells us "As the years passed, it has become more and more evident that many of the hearing disorders of adult life begin in childhood, and if more is to be learned about the causes, treatment and prevention of deafness more studies on children must be made."

Determination of Cause of Deafness—History of the Patient.—The successful treatment and management of deafness depend upon accurate diagnosis and the institution of precise methods of therapy. Accuracy in diagnosis requires a painstaking history embracing not only the previous general and aural ills of the patient but also the family history in so far as it relates to ear disease. Careful inquiry should be made into the patient's habits in regard to eating, health, the use of alcohol, tobacco or narcotics, and the taking of large doses of quinine or similar drugs. The patient should also be questioned regarding the condition of his upper respiratory tract with particular reference to head colds, sore throats, earaches, otorrhea, and allergy. Such points as the incidence, course, and duration of any of the exanthemas or intercurrent respiratory infection should be accurately recorded. The history of traumatism may also be significant. A report upon the careful examination of the teeth, including roentgen-ray examination by an expert, should be obtained in all cases. In chronic deafness, at least a blood Wassermann test is indicated, and it is often desirable, if the former is negative or doubtful, to check up with a spinal fluid test for syphilis, irrespective of the history obtained.

A systemic review for other possible causes for deafness should be undertaken in a comprehensive manner. There may be primary, sec-

ondary, and tertiary causes as well as associated and interrelated factors responsible in any given case. Some of these are the following: cardiovascular-renal disease, pulmonary disease, gastro-intestinal disturbances including hepato-biliary disease, metabolic faults, nutritional imbalance, endocrine dysfunction, blood dyscrasias and deficiencies (either primary or secondary), diseases of the urogenital system, brain tumors, organic lesions of the nervous system (either central or peripheral), and the various endogenous and exogenous toxic states that may arise as a result of adverse hereditary and environmental factors, as well as noisy surroundings, swimming, diving, undue chilling of the body, improper nose blowing, and the irrational efforts of many who attempt to cleanse their external auditory canals of cerumen, or who otherwise believe that they must cleanse the ear canals by introducing soap and water there—a pernicious habit unfortunately practiced by many.

EXAMINATION OF THE PATIENT—In the examination of the ear, slight eversions in the drum membrane, either in color, light reflection, or position, and the state of the external canal wall should be noted and recorded. A normal drum membrane is brilliant, has a pearly luster, reflects the cone of light properly, presents landmarks correctly positioned, and has a normal vibratory power. All of these qualities make for the efficient conduction of vibrations that strike the drum membrane, and if the ossicular chain is free of acute inflammatory processes or has not been impaired by changes resulting from repeated acute inflammatory attacks, or the presence of subacute or chronic states—catarrhal, suppurative, hypertrophic, hyperplastic, or atrophic—the vibrations will be conducted accurately to the intralabyrinthine fluids and from thence to the organ of Corti, to the acoustic nerve and on to the temporal lobe of the opposite side, to the anterior part of the superior whorl, with a few filaments to the same side. Normal hearing presupposes a normal external canal, a normal drum membrane, normal ossicular chain, and an efficient perceptive apparatus, the function of the latter not marred by sensitization, intoxication, inflammation, exhaustion, and the development of permanent, deep-seated morbid processes along the entire path.

Examination of the structures of the upper respiratory tract should be carried out in a

systematic manner in order to evaluate the integrity of these cavities. It must be remembered that each mucous-lined cavity, bony or otherwise, of the head is a potential focus of infection either directly or indirectly, and in searching for such a focus, patience, skill, and mechanical and laboratory aids are essential. No portion must be overlooked and roentgenologic aid is usually necessary, although not to be absolutely relied upon.

The intranasal structures should be examined for changes in the color or appearance of the nasal mucosa, for septal irregularities, spurs, ridges, hypertrophies, hyperplasias, and polyps. The observation of even small quantities of pus or mucopus in the middle and superior meatuses, after the application of negative pressure, may point to a hidden, almost symptomless purulent sinus infection. The use of the postnasal mirror will reveal the state of the posterior tips of the inferior and middle turbinates and show whether or not adenoid masses are present in the epipharynx (the latter are not infrequently found in adults by diligent searching). The introduction of the nasopharyngoscope supplements the study of the nasopharynx and completes it, for the nasopharynx may be considered in many instances as the "font et origo" of ear diseases and dysfunction. With care, one is enabled to view the condition of the pharyngeal orifices of the eustachian tubes, to determine whether or not lymphoid elements are present in and around Rosenmüller's fossae and how much inflammation, infection, secretion, and fibrous bands and adhesions are present, and at the same time to rule out infiltrations, degenerations, nasopharyngeal fibromas, or malignant disease.

In children the examination of the nasopharynx is best accomplished by palpation.

Transillumination of the sinuses offers a certain amount of valuable information from the viewpoint of an accurate appraisal of the frontal and maxillary sinuses. Slight differences should be noted and asymmetries borne in mind. It may be found necessary to irrigate the maxillary sinus and if symptoms and findings suggest repeated lavage, then a cytologic and bacteriologic study of the maxillary sinus washings may further aid in the efficient management of clearing up this particular focus in relation to the problem of deafness, as well as reveal an additional element, as, for example, a latent nasal allergy.

Every part of the pharynx and fauces should be examined with care. If tonsils are present their relationship to the aural condition should be accurately established. In a patient with deafness, a history of recurrent tonsillitis and sore throats, with or without fever, in addition to the expression of pus, caseous material accompanied by pus, or liquid exudate from the crypts, with or without persistent enlargement or tenderness of the node at the angle of the jaw (Wood), constitutes an indication for tonsillectomy. Even after a tonsil operation has been performed the fossae should be examined for stumps, tags, regrowths, small crypts concealed behind the pillars or beneath scar tissue, compensatory hypertrophies of the lateral pharyngeal folds, and isolated masses in the post-pharyngeal wall containing crypts filled with presumably infectious material. The lingual tonsil and the infratonsillar lymph nodes, were shown by French, years ago, to be not uncommon foci of infection.

The oropharynx should be thoroughly cleaned of all demonstrable morbid and pathologic tissues. These tissues may not only be primarily infected but may also cause acute infection of the accessory sinuses and prolong chronic infections of these cavities. They are frequently secondarily infected by sinus drainage, causing, in turn, focal infection, either toxic or actual, elsewhere.

The functional capacity of the acoustic apparatus is best studied by the use of the standard methods, *i. e.*, conversational and whispered voice, watch, fork, Weber's, Schwabach's, Rinne's, Galton's whistle, and audiometer tests. These tests not only give important qualitative and quantitative values, but serve as a basis for future tests administered from time to time, so that the aurist will be in a better position to judge the integrity of the acoustic apparatus as well as to know the progress of the disease. Retesting of hearing acuity should be made at definite intervals and should be properly recorded. While the use of the audiometer is satisfactory and important for this purpose and undoubtedly is less subject to variation due to the personal equation of the examiner and to different surrounding noise conditions it is questionable whether the older methods of testing should not also be employed and the results recorded at least occasionally.

Prognosis in Deafness—In most acute and subacute cases in which the family history is not

unfavorable, the prognosis is good, and the anxiety of the patient may be frankly relieved. In chronic and progressive forms of deafness, particularly those well advanced when first examined, the prognosis varies from doubtful to grave or hopeless. In suggesting treatment to a patient it is usually best to state frankly that much (or little) can probably be done to improve the hearing (or only to arrest or retard the progress of the disease), that the aurist's directions as to habits, examinations, regular attendance, and medication must be carried out faithfully, that no definite promise can be given, that full restoration of function is extremely unlikely, and that even in the event of marked improvement taking place the patient should report at intervals of six months or a year for re-examination to determine if impairment has again set in.

Prevention of Deafness—To prevent irreparable damage to the delicate auditory nerve mechanism it is necessary to minimize as far as possible all the conditions that might be responsible. This can be accomplished by further researches into the underlying causes of the deafness that still remain obscure, by closer cooperation between the medical profession, public health authorities, industrial managers, school authorities, social service workers, and the laity, by educational propaganda awakening the minds of the masses to the importance of good hearing and by adequate legislation to secure desired results.

There is little doubt that many, if not most, cases of progressive deafness that become manifest in adult life have their inception in neglected eustachian tube and middle ear inflammations in infancy and childhood. It therefore behooves the pediatrician and aurist to watch carefully and treat all patients with ear complications of the acute exanthemas or infections of the upper respiratory tract, and to keep them under observation until there is reasonable certainty that normal hearing has been established. To accomplish this, the ears of children suffering from such acute infections should be frequently examined and appropriate treatment directed, during the acute stages, to the nose and nasopharynx. This treatment consists in cleansing the nasal passages with warm neutral solutions introduced with a medicine dropper, and the instillation of shrinking solutions (such as ephedrine) and antiseptic solutions (such as 2 per cent mercurchrome, 5 to 20 per cent

argyrol, or kindred solution) Instillations of bland oil (either plain or containing ephedrine or menthol and camphor in minute quantities and preferably in the form of a nebula) into the nose are also useful Prevention of repeated attacks of tubal inflammation calls for careful surgical cleaning up of the nasopharynx, particularly in the region of Rosenmüller's fossae, and appropriate treatment of nasal accessory sinus disease, as well as meticulous attention to the child's general health After the acute attack is passed, ventilation of the middle ear should be aided by inflations with the Politzer bag and gentle ear-massage In repeated 'colds in the head,' prophylactic vaccines often have a most beneficial effect, the prevention of these 'colds' usually meaning prevention of loss of hearing at the time as well as later in life

In recent years, otologic opinion has been focused upon the relationship of lymphoid elements in the nasopharynx to child deafness and the latter to adult deafness All agree that a thorough adenoidectomy, if possible, should be performed Guggenbeum states that over 50 per cent of the 18,000,000 deafened persons in the United States owe their affliction to tympanic disease resulting from obstruction of the eustachian tubes in early life He points out that the usual 'blind adenoidectomy' does not remove all the adenoid tissue and contributes toward future deafness He advocates the procedure of "direct adenoidectomy"

Audiometric studies are carried out about three weeks after the adenoidectomy Follow-up studies of patients who had had direct adenoidectomy, indicated an improved nasopharyngeal condition as well as improvement in hearing

The remarkable observations of Crowe, given in the section of this book entitled "Irradiation Treatment of Hyperplastic Lymphoid Tissue in the Nasopharynx" (p 166), deserve special consideration and reflection, for upon the integrity of the nasopharynx, with tube mouths normally ventilated and not blocked or obstructed by lymphoid elements, constrictions, adhesions, or inflammatory states, depends the efficiency of the conducting mechanism

Building Body Resistance—The importance of good mental and physical health and vigor cannot be emphasized too strongly as a powerful aid in the treatment of any type of deafness regardless of the etiologic basis thereof Every local and general method should be utilized to

heighten the powers of immunity Every system in the body should be properly rested and exercised, and every proved hygienic principle carried out, to insure proper balance When dysfunction of any system or systems has been found, thorough management to restore normal function should be instituted without delay or the vicious circle may continue

Care in the selection of a well balanced diet will aid in no small way in increasing the general well-being of the patient Fresh vegetables and greens, cod liver oil or its equivalent, milk, butter, and fruits—all in proper balance—will supply the necessary vitamins A, B, C, and D When indicated, if nutritional faults and vitamin and mineral deficiency are manifest, additional concentrates of these important elements are available Exercises (active and passive), attention to posture, and breathing exercises as well as massage, heliotherapy, and hydrotherapy will enhance physical vigor Adequate sleep and regular periods of rest and relaxation will materially aid the kinetic system so that the energy of the body will be sustained and a high reserve of "electric potential" (Crile) will be created Proper clothing and the avoidance of chill and drafts help to preserve the balance of the circulatory and glandular systems thereby preventing the occurrence of a lowered point of resistance in the body Mental hygiene, too, by promoting poise and calmness, furthers functional vigor and combats physical depression It is the judicious combination of all of the mental and physical health measures that will undoubtedly raise the resistance of the body to the highest possible level, aiding the elimination of recurrent acute or chronic sinus and nasopharyngeal disease, if the latter condition be due to faults of personal hygiene, improper diet, fatigue, and/or mental and nerve strain The measures outlined cannot but help any given case, for they constitute definite principles and grounded fundamentals in any scheme of building up the vital powers

The adjustment of metabolic faults, the promotion of elimination, and the normalizing of faulty blood chemistry are of vital import in achieving the best results The endocrine system should be supported and balanced The elimination of endogenous and exogenous poisons will be of help, in many cases, as will a thorough study of allergy and the institution of precise allergic therapy Climatotherapy may be tried, where possible Though favorable results have

been reported in many cases, change of climate has not always proved to be helpful. The physician should always use discretion in advising patients, making it clear that the change may or may not benefit them.

Deafness in Nonsuppurative Middle Ear Disease—Acute and Subacute Conditions—Deafness due to obstruction of the external auditory canal from cerumen, foreign bodies of many kinds, inflammation of the soft parts, or bony growths can obviously only be relieved by thorough removal of the cause, in which case prompt restoration of hearing may be confidently expected in a majority of uncomplicated cases.

When the impairment is of short duration and is due to drugs, such as quinine, alcohol, or tobacco, recovery should follow removal of the causative factor.

When deafness follows "an acute bead cold" and is due to inflammation of the pharyngeal orifice of the eustachian tube and the tubal lining mucosa, with or without exudate in the tympanum, treatment is directed first to the nose and nasopharynx in order to promote aeration and drainage of the nose, nasal accessory sinuses, and tympanum as well as to remove any accumulated secretion or exudate in any part of the nasopharynx. Topical astringent applications to the mouth of the tube and the middle nasal passageways followed by a nebula of bland oil will aid in restoring the ventilation of the middle ear cavity, and later, when the acute stage of the infection has passed by, gentle inflations of the middle ear with the Politzer bag or eustachian catheter will accelerate the return to normal. Judicious massage with a hand or engine-driven masseur is also of benefit, this procedure tending to restore the normal air equilibrium, to remove the products of inflammation, and to break up beginning adhesive bands of connective tissue in the tympanic cavity.

If these acute attacks recur, a thorough search for the cause should be made and foci of infection in the mouth, fauces, nasopharynx, and nasal accessory sinuses removed. Tonics and alteratives are indicated. Exercise, natural or artificial sunshine, proper personal hygiene and clothing, at times the restriction of sugar and starches, and often the addition of fats to the diet may be prescribed. Sburly's prescription of a wine glassful three times daily of the juice obtained by pressing two raw potatoes,

two raw carrots, two raw tomatoes, two raw beets, and a bunch of celery is often of value in children, particularly in those who suffer from enlargement of the cervical lymph nodes with every cold even after the tonsils and adenoid masses have been removed.

Chronic Conditions (Progressive Deafness Other than Otosclerosis)—Careful hearing tests serve to determine the type of deafness, whether middle ear or internal ear impairment predominates. Whereas formerly middle- and internal ear deafness were sharply divided into conductive and perceptive deafness respectively, the modern tendency is to group both types under the general term of "progressive deafness" which may be defined as that form of deafness consisting of both conduction and perception loss in variable amounts, often with a tendency toward further steady or intermittent loss of hearing. Progressive deafness may be of variable origin, and there may or may not have been a history of previous suppuration. Such cases were formerly designated as those of "mixed deafness."

Preliminary to treatment, a careful survey of all foci of infection and an estimate of the probable effect of these foci on the hearing function must be made.

Treatment is undertaken along the following lines. Badly deviated septa, effectually blocking one or both nasal passages, should be straightened, not only to secure better ventilation and reduction of inflammation in the nasopharyngeal mucosa but also as an aid to the elimination of sinus infection. When indicated aeration and drainage of the sinuses should be improved. First such conservative measures as the application of shrinking solutions and packs, infra red therapy, irrigation, mass or capillary suction, posterior suction, displacement therapy (after the method of Proetz), needle puncture, canalization, middle turbinectomy or trimming, making an infratubal window, and sphenoidostomy should be employed. If, in spite of all of these measures, the infection in the sinuses still remains unabated, then radical operation should be undertaken with the view to removing all infected mucosa so as to eliminate the infected sinuses from consideration as a possible cause of the deafness. Tonsillectomy and the extraction of all teeth that are proved infected, as well as those tested and found devitalized, are sound procedures that will be found helpful.

It is obvious that a damaged perceptive element is not amenable to local treatment other than the removal of the causative factor. When this has been accomplished, if the damage is not irreparable or the case of too long standing, some improvement, at times even a considerable one, may result without other treatment. Some otologists recommend that treatment be discontinued when the causative factor has been removed, others advocate some form of local treatment for patients in whom the eustachian tube is stenosed by a hypertrophic mucous membrane. Simple inflation with the cold air douche gives little permanent improvement in these chronic cases, but, if a hot vapor of iodine, menthol, or camphor, or a combination of these drugs, can be introduced into the tube or middle ear at sufficiently close intervals, some improvement may take place and the distressing tinnitus may be allayed. There are electrical beaters obtainable by which hot air under pressure may be passed over crystals of iodine, menthol, or similar substance, the resulting vapor being driven through the silver catheter into the tube and middle ear. These inflations must not be overdone, and they should always be controlled by the diagnostic tube from the operator's ear to the ear being treated. Treatments should be at semiweekly or even shorter intervals for a period of weeks, depending on whether or not improvement can be noticed.

When constriction or marked stenosis of the tube exists it is necessary to dilate the tube. The heated bougie demonstrated by Simon may well be an aid. At no time must force be applied. After the treatment no inflation should be attempted, and the patient must be cautioned against forcible nose blowing. He should be instructed to blow his nose gently with either one or both nostrils open.

There is little danger of infecting the middle ear by the inflation or the dilation procedures if there is no acute infection present in the nose and throat, and if ordinary attention is given to aseptic technic. Gentle massage of the ear drum with a hand or machine driven masseur is usually gratifying to the patient, as is massage of the external auditory meatus with a cotton-tipped applicator carrying yellow oxide of mercury ointment as practiced by Walter Roberts.

When an atrophic type of middle ear deafness exists, and when the eustachian tube is

patulous and the drum head relaxed, no inflations should ever be attempted. The patient must be cautioned against forceful and improper nose-blowing. Otomassage is contraindicated because of the already too mobile drum membrane.

Shambaugh advocates the occasional insufflation of a pinch of powder made up of one part salicylic acid and four parts boric acid in the widely patulous pharyngeal orifices of the eustachian tubes. This produces a certain amount of inflammatory swelling with consequent reduction of the tube lumen.

The drum head may respond temporarily to various agents used to tighten it, for example, collodion applications, applications of cantharides in collodion, or myringotomy, although these agents may fail and a permanent perforation result. The hearing, however, may improve even if the drum membrane remains perforated.

In the treatment for progressive deafness, high frequency currents may be tried. Hays has a glass electrode shaped like an eustachian catheter. This is wrapped in adhesive to the tip to obviate injury if the glass breaks. This electrode is inserted exactly with the same technic as that used for the catheter, and the current is applied for about five to ten minutes. The treatment is said to decongest the tubal mucosa.

Diathermy applied to the middle ear through the external canal by electrodes may help in bringing about the absorption of exudates and adhesions. Mackenzie has tried diathermy in chronic cases with such satisfactory results in diminishing deafness and tinnitus that he has expressed the feeling that it is worth a further trial.

Röntgen-ray treatment for the restoration of hearing is of doubtful value. Its beneficial action, if any, would seem to be in reducing lymphoid elements about the pharyngeal orifice of the eustachian tube.

Deafness in Chronic Suppuration of the Middle Ear.—Attempts to improve the hearing in patients with chronic suppuration of the middle ear should not be made until the suppuration is under control and the ear dry. Failure to wait for this may lead to increased discharge, renewed activity of granulations, and possible damming back of pus into the recesses of the mastoid process.

When the ear is dry and no granulations are present hearing may often be appreciably im-

proved by the application of an artificial drum to the perforation in the membrana tympani. Commercial drums should not be used. A small disk, slightly larger than the perforation, may be made from glazed paper. The disk is moistened lightly and applied by means of a cotton applicator so that it fits snugly over the entire perforation. The glue in the sizing of the paper holds the patch in apposition to the drum head and may continue so to hold it for weeks, after which it will have to be replaced. Hearing is often much improved and there is seldom enough irritation established to cause renewed suppuration. If the latter occurs the paper patch is loosened by the moisture from the middle ear and soon washed out.

Another device consists in a small piece of cotton—shaped like a ball, cylinder, or cone—which is applied through the perforation to the posterior wall of the middle ear. The cotton acts best as a sound conductor or amplifier when moist. This type of hearing aid, which can hardly be classified as a drum, may be used when there is still a slight amount of moisture in the middle ear, but great care must be exercised as it is often irritating and may readily act as a plug to dam back the discharge. It must be removed at frequent intervals and replaced by clean cotton. When no discharge is present, moisture is supplied by dipping the cotton plug in glycerin. By the use of this device the hearing is often sharpened to a remarkable degree, especially when the cotton plug can be fitted closely down over the stapes or its remnants to the niche of the oval window in patients whose drum membrane and ossicles have been largely destroyed. The cone can be dipped in collodion from the point almost to the base, thus rendering its introduction, as well as extraction, easier for the aurist or patient, the latter can be taught to make and apply these plugs himself, and usually becomes proficient in adjusting them to the exact spots in which they do the most good. The ear should, however, be inspected at regular intervals by the aurist to see that no undue irritation is being caused.

The paper disk and cotton plug, of course, are merely palliative appliances. They serve as hearing aids only and in no sense are to be considered as a means of permanently improving the hearing.

Deafness Due to Otosclerosis—For a discussion of deafness due to otosclerosis and its treatment the reader is referred to the article

entitled "Otosclerosis" (p. 370) and to "Technic of the Lempert Fenestration Operation" (p. 380).

Incurable Deafness.—When a patient with very advanced deafness presents himself, or when all measures for improvement of hearing have been tried without success, it becomes the duty of the aurist to tell the patient that his hearing cannot be improved. Lip reading and the use of hearing aids should be recommended.

In many cases it is wise to resort to a suitable hearing aid before undue deterioration of the acoustic apparatus may have set in, since use of the hearing aid does not militate against hearing improvement and may, in fact, be beneficial through stimulation of the hearing apparatus and hearing centers.

In the advanced cases of deafness the final recourse is to lip reading. Learning to read lips is not difficult, however, a good teacher and long patient application on the part of the patient is required. The aurist should be able to direct his patient to a good teacher and should encourage him to join one of the speech reading clubs or a local branch of the League for the Hard of Hearing where he will find much that will help him to keep his morale and bear his affliction cheerfully.

In some cases it is advisable to continue tube and ear treatments even after all hope of improvement has gone by and the patient has been so informed. For, although hearing may not be improved by treatment, the patient can frequently be made more comfortable by periodic ventilation of the middle ear, being relieved of the "fulness of the head" so often and bitterly complained of, and the distressing tinnitus can often be relieved. In addition, by maintaining close touch with the physician in this manner, the patient can be encouraged, cheered, and helped in many ways.

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WILLIAM GORDON

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removed from his ears, the normal ear is occluded by an assistant, and the same words are then spoken. If the patient claims that he fails to hear the words or numerals which he previously heard through the tubes with the allegedly deafened ear, malingering is established.

Erhard's Test—When the external auditory canal of a normal ear is tightly packed with absorbent cotton, sound waves will still be conducted to a limited degree. A loud ticking watch is wont to be heard from a distance of about one to two meters. The patient's so-called 'deaf' ear is occluded with cotton, and the hearing of the normal ear is tested by gradually moving the watch toward the ear and requesting that the patient count the ticks. The patient's normal hearing ear is then occluded, and the unoccluded supposedly deaf ear is similarly tested. If the patient claims failure to hear the watch under one meter's distance, or the distance at which it should be heard by the occluded normal ear, malingering is suspected.

Chumani Moos Test—This test employs a C2 tuning fork. The vibrating fork is held at equal distances from each ear. The patient may claim that he hears the tone better in the normal ear. The vibrating tuning fork is then placed on the vertex of the skull. Hearing it well in both ears if he is malingering, he will hesitate and finally state that he hears it better in the normal ear. In diseases of the conducting apparatus he should hear it better in the diseased ear. If the normal ear is occluded, a malingerer will state that he does not hear the sound of the fork placed on the vertex of the skull.

PSYCHOSOMATIC FACTORS IN LOSS OF HEARING

Psychogenic losses of hearing fall into three categories: (1) those due to conscious and unconscious malingering, (2) those due to loss of discriminative capacity, and (3) those due to psychogenic and hysterical disorders.

Malingering—In this category the apparent amount of hearing loss is exaggerated as compared to that actually present. It is both consciously and unconsciously presented by the patient who is interested, for example, in the result of a litigation after an accident, or in determinations as to acceptability for military service or degrees of deafness incurred while in military service. For detection of this type of malingering, special tests have been devised. Among these are the binaural stethoscope test, Erhard's test and the Chumani Moos test. All three of these tests are devised for the detection of simulated deafness of the unilateral variety. Simulated bilateral deafness is rarely encountered.

Binaural Stethoscope Test—In this test an ordinary binaural stethoscope is used. The patient is blindfolded. The tubes of the stethoscope are placed in his ears, the one leading to the normal ear is occluded with a hemostat. The bell shaped chest end of the instrument is used as a speaking tube. Into it the examiner speaks in a soft low voice, utilizing test words and numerals. The patient is asked to repeat what he hears. The tubes are

Loss of Discriminative Capacity.—The second type of psychogenic loss of hearing stems from the voluntary or involuntary exclusion (by the patient) of the world of sound. This type is generally found in older persons who seem to have lost the usual discriminative capacity which all normal individuals possess. The faculty of discriminative capacity to hear is dependent, to a wide extent, on the amount of attention which the individual concentrates on particular sound-wave stimuli. Biologically, hearing is the sense which warns the individual. The sound stimuli are able to overcome even inhibitions created by sleep, and reach a person's consciousness even against his will. Motor reflexes are evoked as well, in the voluntary as in the involuntary muscles, sometimes without reaching the person's level of consciousness. These acoustic stimuli are easily in the range of intensity of a normal individual, and discriminative capacity is a matter of experience and memory. When hearing becomes impaired, the individual, especially in later life, finds that his present experience based on hearing losses does not correspond any more to his acoustic experience of former times, and he consequently lessens his attention and decreases the amount of voluntary attention which he formerly used in listening. Thus, he increases further his im-

pairment of hearing. He actually fails to use the amount of bearing he still possesses. Acoustic training is indicated.

The individual born with severe hearing loss, never having developed normal acoustic experiences and memories, actually does not know how much he can hear. He fails to use all his acoustic capacities. To a large extent, he behaves like a deafmute. The marginal area of unused bearing may be brought to his consciousness by acoustic training. Progress is manifest by increased comprehension of speech, and interpretation of noises and in an actual increase in hearing measured by distance.

Psychogenic and Hysterical Disorders—These also produce inhibitory mechanisms which cut off the individual's consciousness from his own perceptions of sound. In these types of cases, the reflex mechanisms of the patient still function, and graphs may be made clearly showing motor reflex responses to sound stimuli, yet the subjects appear unaware that they are hearing sound. The technic for recording and determining whether speech was comprehended or not was developed by Otto Lowenstein. In these cases too, acoustic training and psychiatric therapy have been found helpful in increasing hearing acuity.

SAMUEL J. KOPETZKY

HEARING AIDS

Early Hearing Aids—Hearing aids are of many kinds. Probably the first aid was the hand cupped behind the ear, which gives an appreciable amplification to incoming sounds. Its later development took the shape of a large horn which could be used to magnify outgoing sounds. Alexander the Great is said to have used such a suspended goat horn to call together far distant huntsmen. For compactness, the horn was later curled on itself and used as a speaking trumpet for communications between vessels at sea. It finds its modern counterpart in a cheer leader's megaphone. By placing the ear at the small end of a megaphone otherwise inaudible sounds can be heard. Here began the speaking trumpet and speaking tube first described by Athanasius Kircher in 1673. The

speaking tube still holds a useful place in certain extreme forms of deafness.

Dr. Max A. Goldstein discusses bearing devices in his book entitled "Problems of the Deaf" (Laryngoscope Press) and describes the remarkable collection which he gave to the Central Institute for the Deaf at St. Louis. This collection includes specimens ranging from large and cumbersome horns to devices concealed in a fan or hat or in a lady's hair. One type of device, in the form of a plastic rubber fan, secured amplification by bone conduction, the edge of the fan being held between the teeth (Fig. 226).

Electrical Hearing Aids—The first electric amplifying device is credited to Dr. Ferdinand Alt, an assistant at the Politzer Clinic of Vienna, who conceived and produced it in 1900. In 1902, Miller Reese Hutchinson, of Mobile, first made the Akouphone which later became the Acousticon. C. W. Harper of Boston brought out his oriphone the same year. Since then many manufacturers have placed various types of these devices on the market.

The Telephone Type—The first electrical hearing aids were patterned after the telephone. The diaphragm of the transmitter lies against many minute carbon balls or granules resting loosely in cuplike chambers. As the sound wave moves the diaphragm in or out, it increases or decreases the number of contacts in these granules, and similarly changes the amount of electricity going through the circuit furnished by a connected dry-cell low-voltage battery. This creates Alexander Graham Bell's "undulating" current which passes through the cord to the ear piece receiver. Here a second magnet and diaphragm change the electric current back into an in and out movement of the diaphragm, thus recreating the original sound at the ear end of the system (Fig. 227).

If the sound waves are not magnified enough they can be stepped up still more by an amplifier. This is a combination of receiver and transmitter, introduced into the electrical circuit and using the same elements but on a smaller scale.

The Radio Type—More recently, since the development of the radio, the radio principle has been applied to hearing aids. Here the vacuum tube mechanism takes the place of the carbon granule chamber as the agency for stepping up the sound. At first the apparatus was limited to unwieldy desk or box sets. Then physicists designed and made a midget vacuum

tube Three or four of these in a small, easily portable receiver now make possible the modern vacuum tube hearing aid Each vacuum tube has three components and uses two forms

tween these two components is a grid or gate which is connected with the microphone transmitter Variations of sound pressure on the diaphragm affect the "B" current in the grid

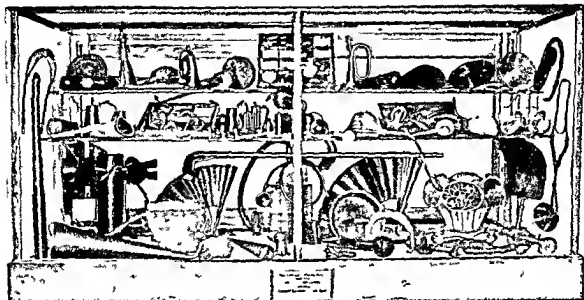


Fig 226 — Photograph of a historic collection of hearing aids

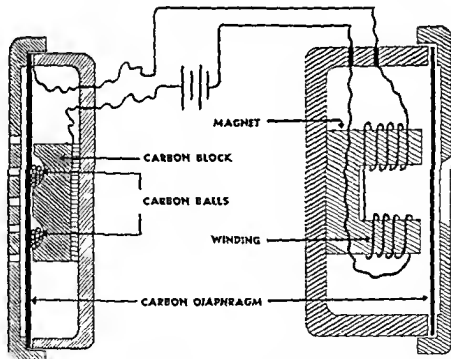


Fig 227 —Diagram of elements of the telephone type of hearing aid, consisting of a transmitter, receiver, battery and connecting cords (From Krantz, Fred W. Hearing Aids Monograph, New York, Sonotone Inc.)

of electric current. A filament is heated by the low voltage "A" battery. The high voltage "B" battery causes an electron stream to pass from this heated filament to a plate. Interposed be-

which acts as a valve or trigger upon the electron stream passing from the heated filament to the gate and thus to the ear piece receiver which is also on the high voltage "B" circuit

(Fig 228) The larger the number of vacuum tubes the greater the trigger action, also the greater the electric battery consumption and the greater the sound amplification. The different shapes and materials used in the carbon or vacuum tube instrument introduce differences in tone and resonance, with peaks of efficiency in certain ranges, and with varying handicaps such as metallic timbre, static, clothing rub, overloading, and leakage squeal.

Sound Frequencies Required in Hearing Aids—The normal hearing range is estimated at 20 to 20,000 cycles while the normal speech sounds range between 90 and 8000 cycles. To hear speech, a range including 500 to 3000 cycles is important while the 1000 to 2000 frequencies are especially essential. The average telephone uses the 300 to 2500 frequencies,

piece receivers, air conduction and bone conduction. The air-conduction terminal is attached to a snugly fitting lucite ear piece (preferably made for the individual) that fills the concha and ear canal down to but not touching the bony portion. The bone-conduction terminal uses a diaphragm that is pressed against the mastoid bone and held there by a spring band over the head. The air conduction ear piece is more efficient, for 30 to 40 decibels of sound is lost in passing through the skull. There are certain patients, however, with very poor air conduction and very good bone conduction, who prefer the latter terminal. Also, if there is an active middle ear or ear canal infection, this will make hazardous the wearing of an air conduction terminal insert, and a bone-conduction terminal is then to be preferred.

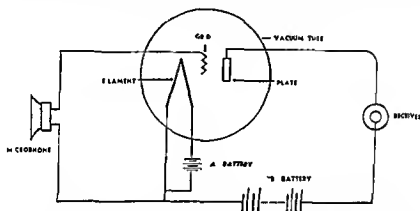


Fig 228—Simplified diagram of the circuit in a vacuum tube amplifier type of hearing aid (From Krantz, Fred W. *Hearing Aids*, Monograph, New York: Sonotone Inc.)

while the average radio covers the 200 to 3500 cycle range. As the softer consonant sounds are in the upper tone range, above 2000 persons with high tone loss have more difficulty in hearing the consonant sounds in speech and are better served by a vacuum-tube aid which enjoys a higher tone range (Fig 229).

In comparing carbon and vacuum tube aids, the carbon aid tends to give a peak amplification in the middle range while the vacuum tube aid maintains a better over all gain and is more efficient in the high tones. In general, a well fitted carbon aid will serve well for a patient with less than a 40 decibel impairment, while a patient with greater impairment will be better served by a vacuum tube aid. Both types of devices are being improved through acoustic research with more emphasis at present on the vacuum tube device.

Ear Receivers—There are two kinds of ear-

The air conduction terminal may be magnetic or crystal in type. The latter gives better performance, especially in the high range, but requires a stronger electric current, hence it serves better in the vacuum tube instrument than in the carbon type.

Fitting Hearing Aids—The selection of the ear to be fitted requires consideration. If the better ear has some useful hearing without amplification (say a 40 decibel loss, or less) and the poorer ear is efficient enough to give a good result with amplification, then the poorer ear is fitted and the patient enjoys good hearing on the amplified side with some useful hearing on the other. If the better ear does not have useful hearing without amplification, then the better ear is fitted because the instrument will not need to be as powerful as it would for the poorer ear and the battery consumption will be less. If both ears are poor and the fitting of one ear

does not seem adequate, the patient will secure more amplification through a binaural fitting. This is said to increase the amplification 7 to 10

divide the different forms of deafness into four groups. In one group there is shown a flat audiometric graph with a fairly even over-all

HUMAN HEARING

SCHEMATIC REPRESENTATION OF INTENSITY AND FREQUENCY CHARACTERISTICS OF THE HUMAN EAR AND LOUDNESS OF SOUNDS

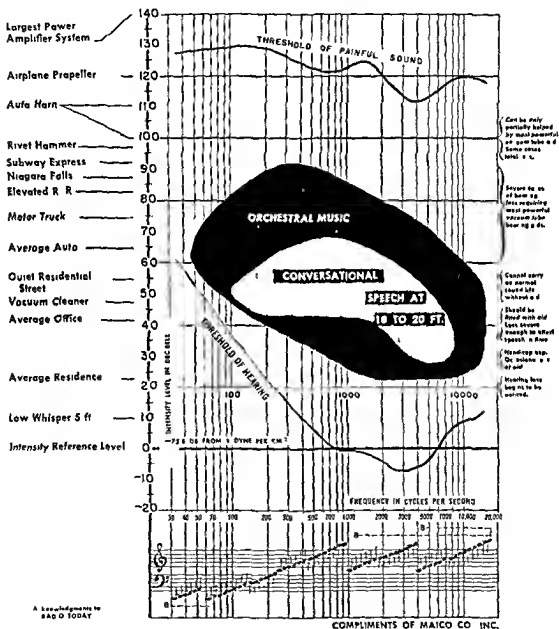


Fig. 229 —Chart illustrating intensity and frequency characteristics of various sounds in relation to human hearing (From Watson L. A. Radio Today, New York, Maico Inc.)

decibels. The relatively few who use binaural aids claim not only more amplification but a better localization of sound.

Some acoustic engineers find it helpful to

loss. In the other three groups, excessive or peak losses in the low, or the middle, or the high frequencies is shown. Through its more level amplification, the vacuum-tube aid lends

itself to a better adjustment in cases in which there is the more marked deafness. The acoustic engineer first steps up all frequencies, then introduces filters to lower the amplification where it is excessive for the individual being fitted. This may be done in the laboratory, or the agent can secure changes through different screw adjustments, or the individual may effect these changes himself through the turning of a control wheel or knob. Having thus secured the desired amplification in the special frequencies the individual then controls the incoming general amplification through a rheostat control of the electric current used.

Efficiency and Benefits of Hearing Aids—A well fitted vacuum tube hearing aid will help those with a loss even as high as 85 decibels, those with hearing loss above this will probably resort to lip reading. The gain to be secured lends itself to mathematical computation. Responses vary, but a good vacuum tube hearing aid should yield at least a 30-decibel gain. Some manufacturers claim gains of as high as 60 decibels but these are probably laboratory results that cannot be secured as yet in practical life. If then the patient has a 50-decibel average loss and can lift this 30 decibels with a hearing aid, he then carries a 20-decibel handicap which gives him enough hearing to manage comfortably under normal social and economic conditions.

The psychological reaction of the patient to a hearing aid is at times startling in its benefit. The acknowledgment of the handicap and the acceptance of the aid is a difficult ordeal for most. Once it has been surmounted, the re-entry into a more normal life may have a profound result on the individual in terms of greater economic assurance, social ease, and self-confidence. Life returns more nearly to normal and the individual responds in satisfaction to himself and in a shared happiness with those whom he contacts.

GORDON BERRY

ACUTE INFECTION OF THE MIDDLE EAR (ACUTE PURULENT OTITIS MEDIA)

Etiology—Acute suppuration of the middle ear follows the introduction of micro organ-

isms into the tympanum. The usual pathway is through the eustachian tube. Other pathways are through the external auditory meatus, through the internal auditory meatus, and through the lymphatics or blood vessels on rare occasions.

Bacteria are the main exciting cause, although other types of micro organisms, such as the fungi, may occasionally be found. Among the bacterial groups, the streptococci, staphylococci, and pneumococci are responsible for the great majority of pyogenic infections. Occasionally we may find *Streptococcus viridans*, *Bacillus proteus*, *Corynebacterium hoffmanni*, *Bacillus influenzae*, *Bacillus tuberculosis*, or others.

The percentages of various organisms found in 200 consecutive cultures at the University of Virginia Hospital,* in Charlottesville, were streptococci 54 per cent, staphylococci 24 per cent, pneumococci 8 per cent, proteus 8 per cent, hoffmanni 4 per cent, and diphtheroids 2 per cent. These percentages vary somewhat from season to season and in various sections of the country. However, the major percent age is always composed of the three main groups as listed above.

The immediate predisposing causes are the inflammatory reactions set up in adjacent regions by virus and bacterial action, as in the common cold, acute and chronic sinusitis, meningitis, external otitis, and in the infectious diseases. Among the remote predisposing causes are certain mechanical factors, such as malformations of the nasal septum and turbinates and a hypertrophied adenoid, which tend to interfere with normal nasal aeration and drainage. Diseased tonsils, general systemic disease, and metabolic disorders may also at times be contributing factors.

Anatomic variations of the eustachian tube and nasopharynx also play a role, for the eustachian tube in infants and young children, as compared with that in older children and adults, is shorter, more patent, and more nearly horizontal, and it is in this age group that middle ear infections are most commonly seen. If the tympanic membrane of any lustily crying infant is observed, marked excursions of the drum head will be noted from the rapidly changing atmospheric pressure. It has also been noted that certain families are more prone to ear infections than others. This has been explained by the inheritance of a certain anatomic pattern

which facilitates the entrance of bacteria into the ear. In infants, improper bottle feeding may be the cause, in older children and adults improper and too forceful blowing of the nose is often responsible. When there is a dry perforation of the drum head from an old infection or injury, or when there is a traumatic rupture from penetrating foreign bodies, concussion, or fracture of the temporal bone, the middle ear is particularly vulnerable because of this direct pathway for the entrance of bacteria.

Pathology.—Whenever we see an infection of the middle ear, by means of a spread through the eustachian tube, we may be sure a certain sequence of events has taken place, variable in duration and intensity of each phase. These events are usually at first catarrhal in nature that is, tubal catarrh or salpingitis, then tubo tympanitis, and finally *acute catarrhal otitis media*. When bacteria are added the inflammatory process becomes purulent. Pathologically, these catarrhal processes, initiated by infection in and about the nose and nasopharynx, cause venous stasis and a transudation of fluid elements from the blood through the vessel walls into the tissue. This edema of the eustachian tube interferes with the action of its ciliated mucosa and also prevents a free interchange of air from the middle ear to the nasopharynx. This condition causes a partial vacuum in the tympanum by the absorption of its residual air, and the rate of transudation of fluid is increased, the *membrana tympani* is retracted, and vascular engorgement of the blood vessels along the malleus and periphery can be seen, the normal luster of the drum head disappears and quite often fluid can be noted in the middle ear.

When bacteria are added at any phase of this catarrhal process, the inflammatory changes become rapidly more marked, and there is an exudation of cellular elements from the blood through the vessel walls. Gas is often found at this stage. Destructive changes of tissue soon follow and progress until drainage is established. Red blood cells are often present in the exudate, and hemorrhagic areas are frequently noted. Edema is marked and often blebs are found on the drum head containing a sero-sanguineous fluid under the elevated epithelial layers. The pressure of the exudative fluid and gas in the middle ear pushes the drum head, as a whole, outward. Areas of exudate are frequently noted on the outer surface. The patho-

logic picture varies, of course, depending on the stage observed, the type and virulence of the infecting organism, the general condition of the patient, and the degree of drainage, if any, through the eustachian tube.

Symptoms.—The general symptoms of acute suppurative otitis media are often masked by the symptoms of the predisposing infectious disease, such as acute rhinitis, acute pharyngitis or tonsillitis, measles, scarlet fever, or meningitis. However, this invasion of the tympanum usually produces an aggravation of the general symptoms of fever, headache, malaise, and anorexia, or nausea and vomiting may appear.

The general symptoms of middle ear disease are particularly significant in infants and young children, for food refusal, nausea and vomiting, and perhaps diarrhea are frequently encountered. It may be said here that the symptoms are pediatric but the signs are otologic. The temperature in children may be very high, 104° F is not uncommon, and in such cases convulsions may appear. A mild leukocytosis is usually present.

The local symptoms direct attention to the ear quite early, except in infants, in whom head rolling, tugging at the ear, and increased fretfulness may be the sole local indications of the disease. A sense of fulness or deafness in the ear, often accompanied by a mild tinnitus, is the earliest symptom. Pain soon follows, and may be described as deep-seated or boring in quality. Pain, like temperature and all other symptoms, varies widely in different cases, depending on many factors, such as intermittent drainage through the eustachian tube, the type and virulence of the organism, and the temperament of the patient.

The appearance of the drum head is the most important *objective*, as well as diagnostic, sign, and examination of the ear should never be omitted in any physical examination, particularly in infants and young children. In order to make this examination certain conditions must be fulfilled. First, magnification and a brilliant source of illumination must be provided, with suitable ear specula, the modern electric otoscope fulfills all these requirements. Second, the examiner must cleanse the ear canal of all cerumen or foreign material, in order to secure a clear image. This may be done by cotton tipped applicators, hooks, curets, or similar instruments provided one is skilful in their use,

otherwise, irrigation with sterile water at body temperature should be done.

The image of the drum head in acute otitis media varies widely. The classical picture is that of a bulging, hyperemic membrane frequently showing areas of hemorrhage and exudate on the surface. At the other extreme, the membrane may be only slightly bulging and so nearly normal in color and thickness that the retained pus in the middle ear can be seen *through the drum head*. Other pictures are those in which there is very little inflammatory swelling within the middle ear, but large bullae are found on the drum head. When these are ruptured by brushing with a cotton tipped applicator, it may be noted that the drum head itself is in approximately a normal position. At other times, owing to the necrotizing action of certain bacteria, the drum head may be rapidly destroyed, and large perforations are found. Again the drum head may be gray in color and markedly thickened.

The recognition of middle ear disease calls for careful study, and even with long experience one is often unable to determine the exact nature of the inflammatory process. Extreme gentleness should be employed in all manipulations, and when necessary in young children, the use of a restraining sheet or anesthetic may be indicated.

Diagnosis.—The diagnosis is usually readily made, however, there are times when it is quite difficult. This is particularly true in cases of external otitis or furunculosis, when one is unable to visualize the drum head because of swelling in the canal. In these cases, one must be guided by the history, the symptoms, and the appearance of the canal, however, a small speculum can usually be gently inserted to a depth sufficient to see a small area of drum head.

Myringitis bullosa is often confusing, especially to the beginner. But here again, the history, the symptoms, and the mechanical rupturing of the bullae with cotton tipped applicators or probes help to differentiate the two conditions.

The differentiation of middle ear disease from otalgia due to reflex pain should not be difficult as long as the drum head can be visualized. Atresia of the canal, unsuspected foreign bodies, and bony exostoses may at times make the diagnosis difficult, especially when the patient has an associated acute rhinitis.

Complications and Sequelae.—When infec-

tion is once present in the middle ear, numerous complications are possible. Among these are mastoiditis, petrositis, meningitis, paralysis of the seventh nerve, jugular bulb thrombosis, and chronic otitis media with permanent perforation of the drum membrane. Among the remote complications are endocarditis, nephritis, and metastatic abscesses, as examples.

The most common sequela to middle ear infections, outside of the usual acute complications, is chronic purulent otitis media, hence, the importance of early diagnosis and treatment. This unfortunate sequela usually arises from neglect of the acute phase of the disease, and the destructive, inflammatory process progresses to such a point that resolution is impossible, and permanent tissue damage takes place, such as destruction of the drum membrane and other structures in the middle ear. In some instances, however, the necrotizing toxins of certain virulent bacteria will destroy the drum head and damage the contents of the middle ear to such an extent that resolution cannot take place, in spite of early and persistent treatment, and a chronic purulent otitis media will be established.

Treatment.—Prophylaxis.—The best prophylaxis of middle ear disease is early treatment of the predisposing condition, whenever possible. In diseases such as acute rhinitis, acute sinusitis, and meningitis, prompt therapy with one of the sulfonamides is indicated. In conditions such as acute rhinitis, the routine use of sulfonamide therapy as a means of preventing complications has not yet proven expedient. Perhaps the day is not far distant, however, when penicillin or some similar chemotherapeutic agent will be used as a routine measure and will considerably reduce complications from both virus and bacterial infections.

The practitioner, in treating acute or chronic so-called *catarrhal otitis media* dependent upon tubal obstruction, should always have in mind the risk of forcing infective material into the middle ear.

Patients should be instructed in the proper method of blowing the nose, the use of nasal douches, and the protection of the ears while swimming.

Finally, the removal of diseased and hypertrophied tonsils and adenoids, the correction of deformities of the septum and turbinates, and treatment of chronically diseased sinuses will materially reduce the incidence of ear infections.

Medical Care and Management—Bed rest and therapy directed towards improvement or cure of the predisposing condition are of the utmost importance. As a rule, it is not necessary to alter the patient's diet, provided it is adequate. Dehydration must be watched for and parenteral fluids given, if necessary. Pain and general malaise should be controlled by one of the coal tar derivatives or codeine, when indicated.

Application of heat to the ear, by means of an electric pad or hot water bottle, often relieves pain. Also, the use of ear drops will diminish the pain and often abort an early infection. Ten per cent phenol in glycerin has been used many years with good results. The proprietary preparation "Auralgan," which is now

the number of mastoidectomies all over the world. This reduction in the complications of middle ear disease may be still further advanced by the addition of penicillin and other newer chemotherapeutic drugs to our armamentarium.

Surgical Treatment—The only operative procedure undertaken for acute purulent otitis media is paracentesis or myringotomy of the tympanic membrane. This procedure should always be carried out as soon as one notes the increasing accumulation of gas and pus in the tympanum, which, of course, is manifested by bulging of the drum membrane. It is better to err on the side of opening an occasional drum head unnecessarily than not to open one when it was necessary.

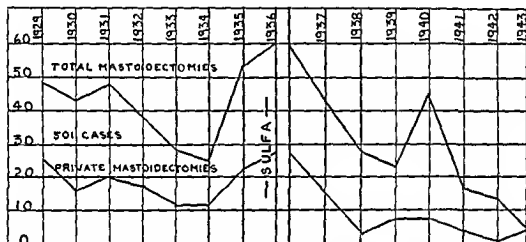


Fig. 230—A graph showing the decrease in the incidence of surgical mastoiditis since the introduction of the sulfonamide drugs (in 1936) in the University of Virginia Hospital at Charlottesville, Va.

so widely employed, is likewise very effective. Using either preparation, it is well to have the ear filled with the medicament every two hours, allowing it to remain in the ear for twenty minutes before lightly plugging the ear with cotton.

When the middle ear has once become involved, the proper sulfonamide drug in adequate dosage should be given and the dosage maintained for from two to four days. Sodium bicarbonate is given at the same time, and if the sulfonamide is given for more than two days, the urine should be examined and the blood level of the drug determined. If resolution does not soon take place, the course of treatment may be repeated in several days. The routine employment of the sulfonamide drugs in these cases has undoubtedly materially lessened

Before incising the drum membrane the ear canal should be properly cleaned. An electric otoscope and a straight handled, sharp myringotomy knife with a small blade are all that is needed, other than applicators and cotton, in the line of instruments or equipment. No anesthetic, either local or general, is necessary in small infants, in older children and adults, either local or general anesthesia may be employed.

For local anesthesia, the instillation of a phenol menthol cocaine solution has proven most satisfactory in my hands. This preparation is made by mixing 30 grains (2 gm) each of phenol and menthol crystals (which immediately go into solution) and adding 30 grains (2 gm) of cocaine (small, fluffy crystals). The ear canal is filled with this solution which is

allowed to remain in the canal for twenty minutes. It is then carefully removed by cotton-tipped applicators. Sufficient anesthesia will have been obtained to make the operation painless. No bad effects from the use of this preparation have been observed in many hundreds of cases. As an extra precaution, the patient's head and hands are held by an attendant, or, if necessary, a restraining sheet is used.

General anesthesia may be used if desired. Any short acting anesthetic, such as nitrous oxide, chloroform, ethyl chloride, or pentothal sodium (administered intravenously), may be employed, depending on the circumstances and facilities available.

The membrana tympani is preferably incised in the posterior half, the incision being made from above downward, following the contour of the canal. Pus and gas promptly gush through the opening. Cultures should be obtained from this material, preferably by dipping the knife blade into a broth culture medium. A knowledge as to the type of organism present is most important in choosing the proper chemotherapeutic drug and in anticipation of certain complications.

A short, clean incision of the drum membrane only is to be desired, for otherwise other structures in the middle ear may be damaged. If the incus is dislocated, a suppurative labyrinthitis and meningitis will soon follow. The facial nerve may be cut or the jugular bulb may be incised in those cases of dehiscences in the floor of the tympanum.

Postoperative Care—In addition to the medical treatment previously outlined, the patient or attendant is instructed to keep the ear wiped out with cotton tipped applicators, the canal lightly plugged with cotton, and the skin greased with cold cream or petrolatum jelly. No irrigations are employed unless the discharge becomes too thick to drain properly, in this case, a sterile salicylic or boric acid solution is used when necessary.

A suppurative maxillary sinusitis is frequently found on the same side as the acute otitis media. When this complication is present, it is very important to irrigate the sinus through the natural ostium, for resolution of the ear infection will not begin until the sinus condition has improved. In children, a persistent otitic discharge will frequently clear up promptly after the surgical removal of adenoids.

The aspiration of thick and tenacious secre-

tions through the perforation in the drum head by means of a fine suction tip is another very useful procedure. At other times, inflation of the eustachian tube will serve to cleanse the middle ear and hasten resolution.

The local use of the sulfonamides (either dry or in suspension) has been disappointing. Iodine (1 or 2 per cent) in boric acid powder insufflated into the canal at the completion of each treatment has been helpful. Perhaps penicillin or some other new chemotherapeutic drug which is effective in the presence of pus will prove of greater value in the future.

Prognosis—The prognosis as to life is very good, but one still sees an occasional fulminating case of meningitis follow an acute middle ear infection, particularly when type III pneumococcus is the offending organism.

The prognosis as to function is also very good especially when treatment is directed to the immediate predisposing factors as well as to the middle ear and adjacent structures. When resolution has taken place, attention to remote predisposing factors, such as malformations of the septum and turbinates and excessive lymphoid tissue in the nasopharynx, will do much in the prevention of deafness.

FLETCHER D. WOODWARD

CHRONIC INFECTION OF THE MIDDLE EAR

Chronic infection of the middle ear (otitis media suppurativa chronica or chronic purulent otitis media) may be defined as a chronic infective involvement of the middle ear, sequent to an unarrested acute infection due to the invasion of septic organisms. Since the middle ear includes the eustachian tube, the tympanic cavity, and the mastoid, chronic infection involves an extensive area. Its chief characteristics are deafness, perforation of the tympanic membrane, and a discharge of chronic, intermittent, or remittent nature.

Just as the borderline between the so-called catarrhal or noninfective serous involvement of the middle ear and the actual infection in that area is often difficult to determine, so the transition from an acute infection to a chronic state

often becomes a matter of otologic opinion. The differentiation is even more difficult when subacute exacerbations continue to occur after a spontaneous or surgical perforative drainage has been completely established. Such decision too depends upon how operative-minded the particular otologist may be. Dench¹ described the transition point conservatively in these words: "We may assume that a discharge from the middle ear, which has failed to yield to proper therapeutic measures at the end of three months, constitutes a symptom of a chronic inflammatory process."

The *eustachian tubes* run upward, outward, and backward from the upper outer cornua of the nasopharynx to the lower anterior part of the tympanic cavity, entering just below the canal which lodges the belly of the tensor tympani muscle. The tympanic end is on a higher level than the mouth of the pharyngeal tube. Situated about one-third of the distance from the tympanum to the pharynx is the constricted isthmus which corresponds to the junction of bony and cartilaginous areas, the bony portion being next to the tympanum. When this isthmus is constricted by any acute inflammation the

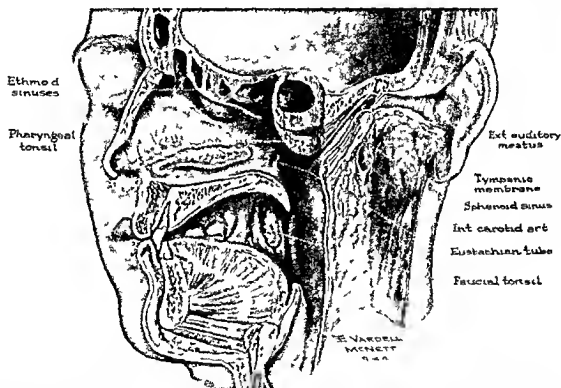


Fig. 231—Schematic illustration of the two passageways by which infections may reach the middle ear: (1) the external auditory canal, (2) the eustachian tube communicating with the air passages. Particles of infected material may traverse these passageways, or infective inflammation may extend by continuity of lining mucosal tissues.

The three divisions of the middle ear are so closely interrelated that involvement of one will probably mean an involvement of one or both of the other two. It is not the intention here to discuss mastoid infection, but the eustachian tube presents such a directly favorable route for sinus and nasopharyngeal infection to reach the tympanic area that it merits a place in the tympanic infective syndrome.

In order to clarify the etiologic and symptomatologic factors of middle ear infection it would seem best to discuss briefly structural phases which contribute to both

air is absorbed by the surrounding blood vessels in the tympanic area and, combined with the external atmospheric pressure from the canal, produces retraction of the drum membrane and constriction of the ossicles and favors erosion of the membrane, the precursor to softening and perforation. On the other hand, by the slower chronic types of closure the same result occurs, i.e., a tubotympanic involvement, and the normal tube function of ventilation and drainage is in degree defeated.

Beneath the mucosa of the cartilaginous part of the tube is the layer of lymphoid tissue called

the "tubal tonsil," which has the same characteristics and susceptibility to infection as the other parts of Waldeyer's ring (the pharyngeal, faucial, and lingual tonsils), as well as the lateral columns of lymphoid tissue behind the posterior tonsil pillars and discrete areas of lymphoid tissue over the general pharynx.

The pharynx and nasopharynx would thus seem to be replete with hyperplastic lymphoid tissue, which is covered by a thin layer of tissue readily eroded and subject to infection from sinus drippings down the postnasal area. The question may arise as to how the infection gets up to the ear when the columnar ciliated mechanism works downward toward the pharynx. In coughing, sneezing, or violent postnasal dis-

posteriorly by way of the iter ad antrum and mastoid antrum with the mastoid region, laterally with the tympanic membrane (directly before the examiner's vision), and mesially with several most important structures. The promontory of the tympanum covers the lower whorl of the cochlea and therefore the area for the highest tones. Above the promontory is the oval window, lodging the footplate of the stapes, below the niche for the round window terminating the scala tympani of the cochlea. Behind the promontory lies the bony roof covering the facial nerve in its canal, upward and farther back, the external semicircular canal.

Here is a perfect opportunity for erosion in most vital areas. The cochlea, facial canal, and

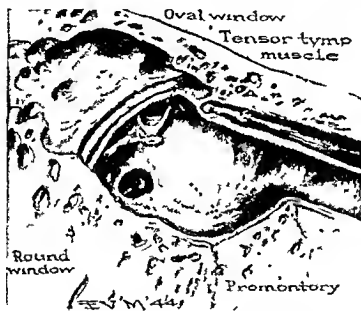


Fig. 232—Mesial wall of middle ear

turbance, infectious organisms and their toxins are readily forced up the eustachian tube, which is of larger diameter in children than in adults, and the tympanic explosion is thus touched off. Careless tubal treatment in the presence of influenza or other upper respiratory infections, the continued necessary packing of the nose for hemorrhage, and improper nasal douching as well as a great variety of other simple conditions would aid the aural transfer of infection. The draining of infected sinus material down through the middle meatus, or farther back from the sphenoid ethmoid recess and sphenoid, aids in bringing about the same result.

The *tympanic cavity* communicates anteriorly with the eustachian tube and nasopharynx,

nonacoustic labyrinth, all must be considered vulnerable in diagnostic study.

While the atrium, or middle division of the tympanic cavity, affords the most generous cavity for collection of posttympanic fluid, with a certain retention gravity collection in the cellular below the attic area, the *attic space* behind the flaccid membrane affords the greatest field for disturbance in general symptomatology. In this area are the heads of the malleus and incus, between which passes the *chorda tympani nerve*, which enters the middle ear by the iter chordae posterius, passes between the neck of the malleus and incus between the mucous and fibrous layers, and leaves by the iter chordae anterius. This nerve carries motor and sensory

fibers joins the lingual branch of the trifacial nerve innervates by secretory fibers the submaxillary and sublingual glands and sends gustatory fibers to the anterior two thirds of the

matic injury The most destructive tympanic invasion is that following diphtheria and scarlet fever measles and influenza are not far behind About 20 per cent of all chronic infections of

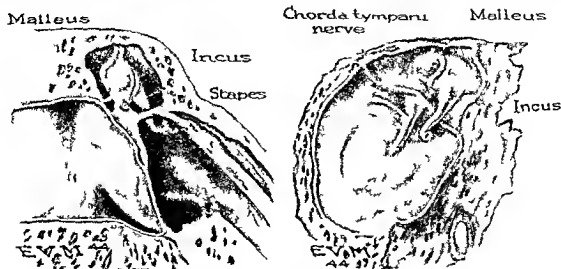


Fig 233 —At the left is an enlarged schematic section of the middle ear and auricular canal At the right a drawing from a dissection showing the tympanum as viewed from within

tongue Its relation to destructive influences in the suppurative middle ear is evident

Etiology —Chronic purulent middle ear infection almost invariably follows one or more

the middle ear would appear primarily due to the infectious diseases of childhood though there may be interim periods of comparative quiet between the first attack and the accept

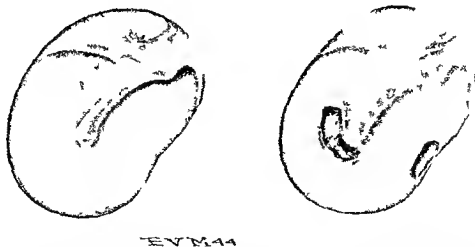


Fig 234 —At the left is a drawing representing a view of the normal tympanic membrane enlarged about 5 diameters At the right is shown two common locations of perforations the central and the lateral

attacks of acute middle ear infection This sequence is not invariable in tuberculous lesions in later stages of syphilis and in the septic invasion from foreign bodies or following trau

ance of diagnosis of chronicity The degree of fixed impairment in hearing is often measured by the destructiveness of the initial invasion

Some ten years ago at the Children's Hospi

tal in Philadelphia Doctors Waltz and Donnelly, in company with the writer, made an effort to determine the exciting factors in chronic middle ear infection from the micro organism standpoint. The results invariably showed a quite mixed infection present. In seventy five children the discharging ears were carefully cleansed and cultures taken through the perforation. The hemolytic streptococcus and *Staphylococcus aureus* were the prevailing organisms. In thirty five of the culture plates single colonies appeared. Twenty two ears were studied by intratympanic culture before opening had occurred from any rupture or paracentesis incision. Here the prevailing organisms listed in the order of frequency were the hemolytic streptococcus, *Staphylococcus aureus* and

trol in the diseases such as scarlet fever, diphtheria, measles, and influenza which have been proven most often basic as etiologic factors including unsuccessful medical and surgical measures to promptly control the acute phase before advanced pathologic change takes place. Into the second group would fall general systemic debility, exhaustion from systemic disease, circulatory imbalance, endocrine deficiency, poor hygienic surroundings (as evidenced by the higher incidence in the poorer sections of the community) and general disorder from the focal standpoint in the upper respiratory tract, nasopharynx, sinuses or other sites.

Reference might be made here to the somewhat exhaustive studies presented by Almour² on the role of squamous epithelium in the cause



Fig. 235.—At the left, multiple perforations of the tympanic membrane. At the right, attic perforations.

Staphylococcus albus. Coincident blood studies showed a marked shift to the left in the Schilling count in ten cases of middle ear suppuration associated with systemic pneumonias in babies who died from the disease.

Since chronic middle ear infection is so frequently a sequence of acute middle ear infection, predisposing factors of the two conditions must in a sense be correlated and would devolve into two important groups. First, those conditions which represent a failure to check the progress and damage being wrought by the acute invasion or failure to terminate its status; and second, those conditions in the individual which represent a poor physiological resistance to the attack. Into the first group would fall the unsuccessful management of the epidemic con-

and repair of chronic suppurative otitis media. Almour discussed the influence first cited by Aschoff³ of the role of otitis media neonatorum due to early respiratory forcing of meconium and other foreign substance into the middle ear, and the role of true suppurative otitis media of infancy due to bacterial invasion through the eustachian tube or by blood and lymph streams from an infected canal in halting the normal pneumatization of the mastoid and in leading to a sclerotic and diploic picture respectively.

Pathology.—The mucous membrane of the tympanic area becomes markedly thickened and hyperemic with areas of round-cell infiltration and formation of granulations, polyps and granulation cushions. New connective tissue

pervades this changed mucosa, there is an in growth of epithelium from the margins of the larger perforations, and the insweep of squamous epithelium from marginal perforations stimulates the formation of cholesteatoma. The small attic perforations, spontaneous in formation, whose favorite sites are above or on either side near the short process of the malleus spell complicating danger in the attic area, in the development of a pressure mass. This dimpling prior to the small attic perforation is a matter of particular concern when occurring over the level of the incudostapedial joint. This promptly extends upward into the attic area with erosion and denudation of bony margins. Then commences the formation of the cholesteatomatous union with superior wall pressure in the neighborhood of the tegmen tympani and tegmen antri causing atrophy in the superior wall—a precursor to bony change and invitation to pathologic change in the adjacent intracranial area. The erosions produced in any bony tissue in the attic area or auricular canal all form part of the pathologic syndrome. Caries and necrosis may affect the ossicles, especially the long process of the incus, in which the circulation is poor. The malleus is also often involved. Rarely, both of these ossicles may be destroyed.

Symptoms—The general symptoms are not proportional to the growing changes from erosion taking place in vital areas on the mesial wall or cranial vault. Pain is usually not present to any marked degree unless the drainage is blocked, some pressure is exerted around the neighborhood of a polyp, ankylosis is present, or there are adhesions around the stapes. Erosion in the walls of the fallopian aqueduct or in any canal adjacent to nerve structure may set up a periostitis with pain from inflamed periotum. Occasionally vertigo is present or tinnitus in variable degree.

Under general symptomatology two neurologic contacts should be emphasized: (1) the symptoms referable to the deterioration or destruction of the chorda tympani nerve and (2) certain subacute expressions of disturbance of the facial (seventh) nerve in its canal so closely adjacent to the tympanic cavity. In the first condition the disturbance in the acuity of taste on the lateral side of the tongue and mouth may be very slight or so pronounced that the patient calls attention to the disturbance and suggests to the otologist, who has doubtless already noted the indifference of the patient to

a long continued otorrhea of subacute character, that something of more significance is going on in the attic area of the tympanum which deserves investigation. This resembles the picture presented when the so called "ear cough" calls attention to the presence of disturbance in the external auricular canal, producing irritation of Arnold's branch of the vagus and through the correlated chain of nerve influence may credit the larynx with its discovery. When there is disturbance of the facial nerve there may not be a definite paralysis but minor symptoms of defective innervation. Careful study of symptoms may here again stimulate a closer examination of erosive action and a suspicion of the existence of damaging dehiscence in the attic area.

The predominant local symptom is that of suppurative discharge and its perforation exit. The discharge may be profuse and constant or so scant that it tends to dry over the drum head quite obscuring a tiny perforation. It may present the mucoid, almost serous, characteristic of secretion from the lower part of the tympanum, or it may be thick and creamy, perhaps with cholesteatomatous debris, suggesting its attic origin. It may have varying degrees of odor, dependent upon obstructed drainage, and it may be stained with blood from sensitive granulations with their own rich blood supply. These varied manifestations influence the treatment to be followed and also explain certain apparently spontaneous recoveries with replacement of denuded tympanic areas by a form of false membrane, thin and fibrous, and chalky spots representing former perforations.

The subjective symptoms of the patient with persistent otorrhea, with or without odor or blood stain and impaired hearing, have already been reviewed. Certain objective symptoms might be cited, such as voice change from fixed deafness, which is probably due to some toxic involvement of the cochlea and limitation of stapedial movement such as would occur in conjunction with erosion of the incudostapedial articulation. This change of voice, due to the patient's inability to hear his own phonation well, would, of course, be more marked in progressive deafness of binaural character. Observation of the patient would show his effort, by posture or other means, to compensate by use of the opposite ear if suppuration is unilateral. Certain minor changes in facial control

might be noted, as well as lack of balance and coordination from labyrinthine involvement.

Diagnosis.—An important differential diagnosis would be the determination of the source of purulent discharge, whether from middle ear or from an old chronic external otitis of the auricular canal. The coexistence of deep exostoses, granuloma, or a severe myringitis with blebs might complicate or clarify the diagnostic picture.

Complications.—The most natural complication of chronic suppurative otitis media is involvement of the mastoid area. This may be sequent to caries from cholesteatomatous pressure in the mastoid antrum. With evident caries in the temporal bone and suspicion of labyrinthine or intracranial symptoms, radical mastoid surgery is indicated. According to Turner,¹ infection may spread, first, medially to the labyrinth through the oval or round windows, or by erosion of the lateral semicircular canal (in rare case, especially tuberculous, the promontory may be eroded), second, upwards through the middle cranial fossa, with extradural or temporal lobe abscess, third, backwards through the posterior cranial fossa, producing (a) an extradural abscess between the sigmoid sinus and its bony wall, or one medial to the sinus, (b) septic thrombosis of the sigmoid portion of the lateral sinus, (c) purulent leptomeningitis beginning in the posterior cranial fossa, (d) cerebellar abscess.

Prognosis.—In a certain number of uncomplicated cases of chronic infection there is a spontaneous recovery with thin membranous covering over portions of the tympanum and perforations. Labyrinthine complications will result favorably or unfavorably according to the success of indicated surgery.

Treatment.—*Prophylaxis and Medical Management*—Prophylaxis should include all therapeutic measures to build up the patient's resistance, to assure integrity of the circulatory system, and to regulate elimination. In every acute infective otitis media there is a potential chronic otitis in the offing. In the interim of uncertainty, scrupulous attention should be given to keeping the external auditory canal in good condition, protective care should be taken of the rhinopharyngeal tract, and effort made to keep the pharyngeal end of eustachian tube open and healthy. I have found sulfadiazine of value during the acute infective stage, but of little value after chronicity is established. Sul-

fathiazole has the advantage of being less soluble, hence remains longer. Recently penicillin has been used with good effect in cases of predominance of organisms sensitive to its bacteriostatic effect.

In definitely established chronic purulent otitis media, it is of first importance to carry out all possible conservative measures. The otologist and the patient must not be complacent about the continuance of the disease in the mild form. The symptoms are classic, the treatment consists first of all in an attempt to clean up the field of infection by every resource of antiseptic measure proving adequate from simple boric acid and alcohol to stronger lotions such as iodine and bichloride solutions. The use of iodine and boric acid powder in the Sulzberger formula (finely powdered iodine, 1 or 2 per cent, in powdered boric acid) has been widely and successfully used, but to be effective must absolutely reach the middle ear and not be deposited in sufficient quantity to prove an irritant. If discharge is offensive, flaky, and tenacious, actual syringing will be necessary, often preceded by peroxide and phenol glycerin, to soften tenacious particles.

In rather quiescent infections, with slight amount of discharge and freedom from odor and pain, we have found that simple pooling of the ear with alkaline antiseptic solutions for several ten minute periods during the day, with the patient resting his head upon the good side, has proved effective. This may be reinforced by the insertion of a gauze wick, moistened with the Randall A. P. C. formula (alcohol 10, phenol 20, camphor 60), deep in the auricular canal, and left over night followed perhaps by a thorough dusting of the iodine boric powder in the morning.

Operative Treatment—The primary indication for operation would be the failure to relieve the progressiveness of the suppurative trouble by careful conservative measures. An equally important indication would be the sudden development of facial nerve, labyrinthine, or mastoid complications.

The operative procedure may vary from simple readjustment and increase of existing tympanic drainage to a radical or modified radical mastoidectomy.

The time-honored operation of ossiculectomy seems to have fallen into disfavor because of the frequent failure to eliminate the chronic discharge. A carefully performed radical mas-

toideotomy would seem to present as good or better prospect in preservation of hearing and halting the infective discharge

A MODIFIED ATTIC DRAINAGE—A careful observation of the picture in the more passive

combining such an opening with any pre existing perforation. Care should be taken not to injure the incudostapedial articulation. All adhesive bands are swept clear, but any free ossicular elements even though eroded, are

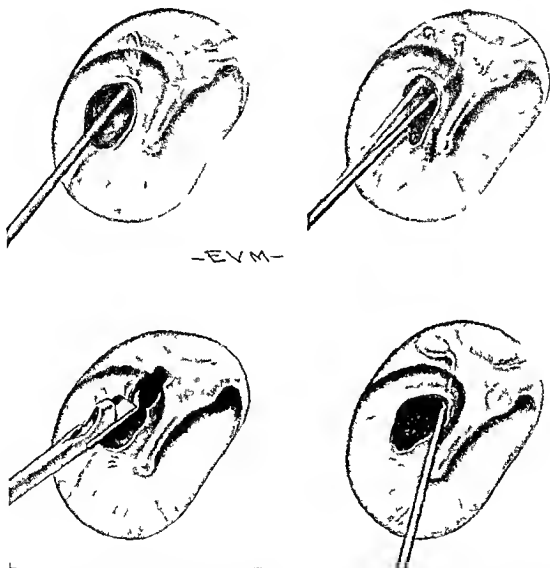


Fig. 236—Enlarged drawings to illustrate the technic of operation for drainage of the attic. At the left above is shown the clearing of adhesions and obstructions to drainage by means of an angular probe inserted through an enlarged tympanic perforation. At the right above is shown two positions of the probe as it is swept sidewise. At the left below is illustrated the method of punching off the margins of a perforation for the double purpose of improving both access and attic drainage. At the right below is demonstrated the method of curettage of the iter ad antrum for the removal of obstructive granulation tissue.

of these chronic suppurative cases has led to the frequent adoption of the method illustrated in Figure 236 which for want of better title is termed a modified attic drainage.¹⁵ This consists in making a free opening in the two posterior quadrants of the tympanic membrane,

left in situ as freedom from pressure will tend to restore their vitality and leave them as a supporting framework to the ossicular chain. This procedure is nothing more than increasing the attic drainage. The denuded areas are usually covered over with a plastic fibrous surface

and hearing often improves. The operation takes little time, can be performed under nitrous oxide and oxygen anesthetic, and requires few instruments and comparatively little subsequent attention. The results have been favorable in over 50 per cent of the cases in which it has been used.

JAMES A. BABBITT

anatomic anomalies, there are differences in symptomatology in association with identical disease processes.

The bacterial flora includes *Streptococcus haemolyticus*, *Streptococcus nonhaemolyticus*, *pneumococcus*, *Staphylococcus nonhaemolyticus*, *Streptococcus viridans*, *Bacillus coli communis*, *Friedlander's bacillus*, and so on. These

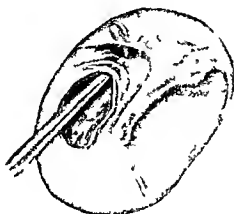


Fig. 237—The drawing at the left shows the first step in modified attic drainage. At the right, a drawing of the appearance of the tympanic membrane after healing.

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MASTOIDITIS AND SINUS PHLEBITIS

General Considerations—The temporal bone occupies a peculiarly strategic position in the skull, and because of its anatomic variations (pneumatic, diploic, and sclerotic) as well as its

micro organisms behave differently in different types of temporal bone. The cellular or pneumatic bone presents demineralization with extensive bony destruction, halisteresis, sequestra, and cavity formation. The pathologic entity is that of coalescence and osteitis. The diploic bone, consisting of bone marrow, frequently is attacked by osteomyelitis, as is often the case in infants and children, or where pneumatization has failed. The sclerotic type of temporal bone is the result of chronic suppuration and is characterized by hard, eburnated, ivory-like bone. This is nature's method of healing and usually results in an osteometaplasia which becomes compact in character. Chronic suppuration may be benign for many years and may remain so unless one of the vital structures, e.g., the lateral sinus, the cerebrum, cerebellum, or labyrinth, becomes involved. Since the bone in this type of mastoid process is eburnated, it is difficult for organisms to attack it directly. However, should the lateral sinus become involved, a phlebitis or a thrombosis may occur. The infection may then spread through the superior and inferior petrosal sinuses, affecting

the cavernous sinus and other intracranial sinuses. Should the meninges become involved, pachymeningitis, cerebral or cerebellar abscess, or diffused meningitis may result.

All the cranial nerves are intimately associated, directly or indirectly, with the temporal bone. The fifth, sixth, seventh, and eighth nerves are most frequently involved, while the

veins, are also closely related to the temporal bone.

To illustrate further the intricacies of the temporal bone it will be recalled that it communicates peripherally through the eustachian tube. The membranocartilaginous portion of the tube terminates at the nose and is constantly exposed to nasal drainage and upper respira-

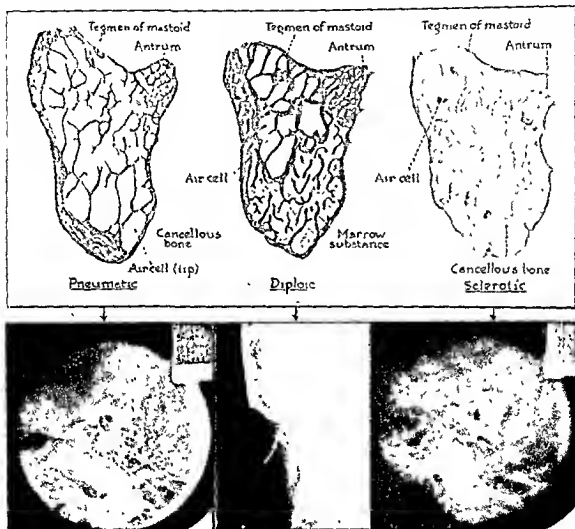


Fig. 238.—Types of mastoid processes. The three types of bone (above) and x-ray appearances of the three varieties (below). (From [above] Lederer and Hollender: *Textbook of the Ear, Nose and Throat*, F. A. Davis Company; and [below] x-ray films, Department of Otolaryngology and Roentgenology, Temple University.)

ninth, tenth, and eleventh nerves become involved only when the jugular foramina participate in the infection. The symptoms may be either motor or sensory.

The important circulatory structures of the head, including the internal carotid artery, the lateral sinus, the superior and inferior petrosal sinuses, the cavernous sinus, the jugular bulb, the internal jugular vein, and many emissary

tory infection. The pathologic processes affecting the mucous membrane of the eustachian tube and the nasal mucous membrane are likely to affect the eustachian tube, and the ear may become secondarily involved. Many infections have their inception in the upper respiratory tract, and the temporal bone may thus become affected either by continuity or through hematogenous or lymphatic channels.

One can readily understand why so many complications with their attendant barrage of symptoms may arise, if due consideration is given the numerous factors involved. These include first the complicated structure of the temporal bone itself, so closely related to the brain and in direct proximity to the cranial nerves, the middle and posterior fossae, the ganglia, and blood vessels. Very few temporal bones are morphologically alike, therefore, each individ-

ACUTE MASTOIDITIS

Acute mastoiditis may be defined as an acute inflammatory disease of the mastoid process and its surrounding structures. An acute suppurative mastoiditis is almost invariably secondary to a suppurative lesion of the middle ear, which, in turn, may be secondary to some upper respiratory infection, such as sinusitis. In children, otitis media may be of exanthematous origin, it often results from measles or

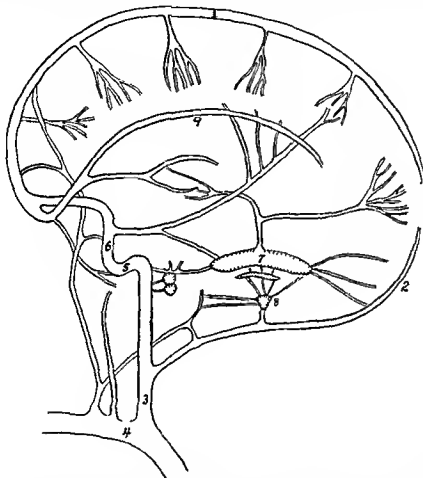


Fig. 239—Sinuses of dura mater showing extracranial communications. 1 Superior longitudinal sinus 2 Branch of facial vein 3 Internal jugular vein 4 Subclavian vein 5 Sigmoid sinus 6 Lateral sinus 7 Cavernous sinus 8 Pterygoid plexus 9 Inferior longitudinal sinus (From original drawing by Dr. B. Chernoff—Class of 1934—Temple University School of Medicine. Submitted as part of a thesis in Otolaryngology.)

ual is predisposed to react to infection in his or her own particular manner.

The diagnosis of an otitic lesion is so different from that of any other surgical lesion. However, we do stress the importance of (1) the history, (2) present illness, (3) subjective symptoms, (4) objective symptoms, (5) general symptoms, (6) laboratory aids (blood count, for example), (7) collateral aids to diagnosis (such as the roentgenogram), and (8) findings at operation.

scarlet fever. Primary mastoiditis due to syphilis or tuberculosis is very rarely encountered.

As has been mentioned, the bacterial flora may be predominantly *Streptococcus haemolyticus*, *Streptococcus nonhaemolyticus*, *pneumococci*, *Streptococcus viridans*, and so on.

The pathologic background may be that of inflammation, congestion, round-cell infiltration, exudation, halisteresis, suppuration, granulation tissue, coalescence, cavity formation, sequestration, demineralization, thrombosis of

the small blood vessels, osteitis in pneumatized bone, and osteomyelitis in diploic bone

Types of Acute Mastoiditis.—From a symptomatic standpoint mastoiditis may be classified as follows

A Typical mastoiditis

- 1 Acute or fulminating type
- 2 Perforative type resulting in
 - (a) Postauricular or subperiosteal abscess
 - (b) Bezold's abscess
 - (c) Zygomatic perforation

B Atypical mastoiditis

- 1 Primary mastoiditis
- 2 Diabetic mastoiditis
- 3 Type III pneumococcus

In either the typical or atypical forms the pathologic background is that of congestion, exudation, thrombosis, coalescence, osteitis, or osteomyelitis, singly or in combination

Typical Mastoiditis—**ACUTE OR FULMINATING TYPE**—In mastoiditis of this type, pain may be sudden in onset and deep seated or localized on the side of the mastoid involved. Occasionally it is distributed over other roots of the fifth nerve. It may be continuous or intermittent, usually becoming worse at night. Fever may be remittent, intermittent, or sustained, and may subside with the institution of drainage. If elevated temperature is persistent, it may be due to toxic absorption or venous phlebitis in the mastoid region, to necrosis of bone, or to tissue destruction. Absence of fever is no indication that the mastoid process is not involved. There may be physical unrest, lassitude, and anorexia. In children, cervical adenitis is a common concomitant occurrence. *Nervous symptoms* are very likely to appear, and may manifest themselves in the form of convulsions, vomiting, vertigo, and occasional suggestive Kernig's and Babinski's signs. *Gastro intestinal symptoms* are often noted but may not necessarily be caused by mastoiditis.

Otorrhea, or aural discharge, is not a constant factor. Some patients may have a profuse and copious discharge, this is a typical finding in coalescent mastoiditis and occurs in the pneumatized mastoid when there is rapid demineralization with extensive mucous membrane and bony destruction. Other patients may have a scanty discharge of a serosanguineous consistency, this usually occurs in conjunction with hemorrhagic mastoiditis when the *Strepto-*

coccus haemolyticus is the causative organism. Cessation of the aural discharge does not necessarily indicate an amelioration of the condition. It may merely signify the presence of a mechanical factor, such as an obstruction of drainage between the middle ear and the mastoid. The fact that the middle ear may be undergoing resolution is not to be regarded as a safe indication that the disease in the mastoid is not progressing.

Inspection of the external auditory canal may reveal a swelling and narrowing of the lumen of the canal with a slight bulging or edema in the posterosuperior wall of the external osseous meatus. The membranocartilaginous portion of the canal often becomes distorted secondary to the edema in the osseous canal. This is a constant factor when there is perforation of the cortex with auricular displacement.

Upon inspection of the drum head, one may note redness and swelling due to a localized ballooning of the weakest portion of the membrane. As a rule the position of the drum head is distorted. A frequent finding is bulging of the posterosuperior quadrant. In typical acute mastoiditis the tympanic membrane is usually dark red, however, the purulent contents of the tympanic cavity may give it a grayish ashen appearance.

Tympanic perforations may be small or large, single or multiple. The location of a spontaneous perforation may be anywhere in the tympanic membrane but its site is of diagnostic value. For example, a perforation in Shrapnell's membrane is significant of a pathologic process in the epitympanum, a perforation along the floor is suggestive of necrotic bone over the jugular bulb, a perforation along the posterior quadrant is due to bony necrosis in the mastoid.

On postauricular and periauricular inspection, absence or partial obliteration of the sulci may be noted. This is a symptom of considerable importance. The sulci are obliterated in infections of the mastoid when periostitis is present and in association with postauricular edema due to a perforation of the cortex.

In a diseased mastoid, on palpation, hyperesthesia localized over the mastoid process is noted. Tenderness may be elicited either by superficial or deep palpation and the degree of mastoid tenderness naturally depends upon the type of bone involved. For example, a mastoid of thin cortex will give rise to greater tenderness upon palpation than a sclerotic mastoid. Three

classical points of tenderness noted in acute mastoiditis are the antrum, the emissary vein, and the area over the tip

Conductive deafness is a typical finding in acute mastoiditis. There is a loss in low-tone perception and the patient's ability to hear whispered and spoken voices is reduced



Fig 240—Classical points of tenderness on palpation
1, antrum 2, emissary vein 3 tip

Blood studies often reveal a secondary anemia and a leukocytosis

PERFORATIVE TYPE OF ACUTE MASTOIDITIS—Mastoid perforations occur whenever there is bone destruction. In this condition there is a break in the cortex with an escape of pus through the fistula. In infants, in whom the bones are not fully ossified, the pus makes its escape through the squamomastoid suture. Perforation in the mastoid cortex may be due to localized halisteresis, increased localized intramastoid pressure, and large cells in the tip in patients in whom the bony plate is thin.

A perforation through the lateral wall will develop into a localized subperiosteal abscess. When the mastoid tip happens to be very thin at the inner medial wall and when the pus escapes through the digastric fossa, a Bezold's

abscess may result. A perforation through the anterior wall will empty itself through the external auditory canal. Edema occurring in the anterosuperior auricular region is usually due to a perforation in the zygoma. Downward perforations empty themselves either through the digastric fossa or through the outer portion of the mastoid tip at the attachment of the sternocleidomastoid muscle.

Subperiosteal Abscess—There are certain auricular displacements and changes in the outline of the soft parts about the ear which may lead one to suspect mastoiditis with a perforation. In adults such auricular displacement is infrequent, but in children it is a rather common occurrence. The abscess is the result of a perforation in the mastoid cortex. The pus makes its escape through the perforation and elevates the periosteum from the surrounding bone.



Fig 241—Subperiosteal abscess.

If the perforation is behind the auricular attachment, the auricle is pushed forward and protrudes rather prominently from the side of the head. If the perforation is at the higher level, the auricle is pushed forward and protrudes rather prominently outward from the side of the head. If the perforation is at a still higher level, the auricle is displaced forward and downward.

As a point for differential diagnosis, attention is called to a similar deformity which is often found in association with furunculosis of the external auditory canal

Bezold's Abscess—This occurs when the tip cells are large, and the bony plate forming the medial wall of the tip is quite thin. The pus follows the line of least resistance and makes its escape through the lower portion of the mastoid into the neck, through the digastric fossa be-

men. Postauricular examination reveals the obliteration of the sulci. Tenderness is most marked at the inner portion of the mastoid tip.

Zygomatic Perforation—The first indication of zygomatic perforation is swelling over the temporal muscle around the zygomatic region which gradually moves downward anteriorly opposite the tragus. The swelling often resembles that of mumps and it is not uncommon to find this type of perforation in children. The

DIFFERENTIAL DIAGNOSIS BETWEEN FURUNCULOSIS OF THE EXTERNAL AUDITORY CANAL AND SUBPERIOSTEAL MASTOID ABSCESS

Subperiosteal Mastoid Abscess

General Symptoms

- 1 Pain—intermittent and nocturnal. Induced by palpation over the antrum, tip and emissary vein
- 2 Temperature ranging from 99° to 103° F
- 3 Patient toxic and ill
- 4 Condition due to an underlying infection
- 5 History of otitis media
- 6 History of otitis media

Furunculosis

- 1 Pain—intermittent. Induced by application of pressure over the tragus, by manipulation of the auricle, by pressure applied midway between the angle of the jaw and mastoid tip, by mastication
- 2 Temperature normal
- 3 Patient fatigued owing to loss of sleep
- 4 No underlying infection
- 5 Onset sudden
- 6 Recurrent with history of traumatism or history of furunculosis

External Auditory Canal

- | | |
|---|---------------------------------|
| 7 Lumen of membranous portion wide open | 7 Lumen obliterated and swollen |
| 8 Infection not localized | 8 Infection localized |

Osseous Canal

- | | |
|---|---------------------|
| 9 Narrowing with edema in the posterior superior quadrant | 9 Normal appearance |
|---|---------------------|

Drum Head

- | | |
|--|--|
| 10 Bulging with perforation | 10 No bulging |
| 11 Suppuration—which may be profuse thin or mucoid | 11 Suppuration—scanty thick and corelike |

Roentgen Ray Findings

- | | |
|---|--|
| 12 Positive pathologic changes noted on roentgen ray film | 12 Roentgenograms showing no disease process |
|---|--|

Blood Studies

- | | |
|----------------------------|--------------------------|
| 13 Leukocytosis and anemia | 13 No associated changes |
|----------------------------|--------------------------|

Bacteriological Findings

- | | |
|-----------------|------------------|
| 14 Streptococci | 14 Staphylococci |
|-----------------|------------------|

neath the sternocleidomastoid muscle. Bezold's abscess is usually accompanied by high temperature. One observes a large mass extending from the tip of the mastoid along the angle of the jaw underneath the mandible. It is often mistaken for Ludwig's angina despite the fact that the location is not typical. Facial palsy is frequently concomitant with Bezold's abscess. This is because there is pressure upon the facial nerve in its exit through the stylomastoid foramen.

temperature is the typical septic type ranging from 99° to 103° F. The history will reveal a stormy siege of otitis media with delayed middle ear drainage. On percussion one will find localized pain over the temporal region and scalp tenderness.

Atypical Mastoiditis—In atypical mastoiditis we find a perverted clinical picture absolutely devoid of the syndrome that characterizes the typical case of mastoiditis. For example it

is not uncommon to find a normal temperature no pain and no aural suppuration. The patient has some vague symptoms referable to the ear and these if properly interpreted will lead to the correct diagnosis. We shall consider under atypical mastoiditis (1) primary mastoiditis (2) diabetic mastoiditis and (3) mastoiditis due to type III pneumococcus.



Fig. 242—Bezold's abscess

PRIMARY MASTOIDITIS—Primary mastoiditis is a pathologic entity secondary to tympanic involvement. Otitis media may be absent. The symptoms are mild and fleeting.

In one type of primary mastoiditis there is postauricular edema and auricular displacement without otitic symptoms. The symptoms of otitis media are usually so mild and fleeting that they pass unnoticed by the patient. The tympanic examination usually reveals no abnormality and there are no changes in the normal landmarks. The infection may have taken place through the hematogenous route from the middle ear into the mastoid with middle ear resolution apparently taking place while the mastoid infection continues. Occasionally there is obstruction in the aditus ad antrum completely sealing off the communication between the tympanic cavity and the mastoid. The middle ear infection subsides while the mastoid destruction continues. The first indication of otitic disease is the discovery of postauricular edema. This type of infection is particularly prevalent in children.

In a second type there are mild middle ear symptoms without mastoid phenomena. The patient may complain of a dull, inconstant pain in the ear. No definite tenderness is elicited but on deep palpation one notes localized areas of tenderness. The drumhead is lusterless and at times one finds a fullness in the posterosuperior meatal wall. The latter is the only positive finding which indicates the diagnosis. A characteristic feature common to both conditions is the absence of premonitory middle ear symptoms. Both types give rise to a slight impairment in hearing which is of the conductive variety and in both forms there is an absence of the typical mastoid pain.

In a third type there are fulminating toxic symptoms, the focus of the infection being in the mastoid but there are no symptoms referable to the ear. The patient presents only toxic symptoms consisting of elevated temperature, headache and general septic phenomena. In this third group also belong middle ear infections associated with fleeting symptoms. Apparently the mastoid becomes involved through the



Fig. 243—Zygomatic perforation

hematogenous or lymphogenous route and the infection is carried into distant areas of the temporal bone. Here also there is no history of aural involvement.

DIABETIC MASTOIDITIS—Acute mastoiditis in diabetics in whom the diabetes is not controlled with insulin belongs to the atypical group because in most instances there is an absence of

pain and temperature. The most outstanding symptom is profuse suppuration. Some clinicians explain the absence of pain as a result of rapid halteresis and anesthesia of the sensory terminal nerve endings. During the early stages of the disease one often sees a complete melting away of the tympanic membrane with rapid cavitation in the mastoid. The diabetic patient with mastoiditis may not have the typical mastoid pain, but does have pain and headaches. The pain is due to a toxid or a diabetic neuritis. The cause of the headache is generally confirmed at operation when one invariably finds an epidural or perisinuous abscess. Symptoms of acidosis may occur and at times becloud the otitic clinical picture. In addition the patient may have an impairment in hearing which appears early and is of the conductive type. As the mastoid destruction progresses the deafness becomes more marked and gradually develops into the perceptive type.

Mastoiditis developing in diabetics in whom the diabetes is controlled with insulin runs a typical course. When it is difficult to reduce the hyperglycemia and the glycosuria the course of mastoiditis is atypical.

MASTOIDITIS DUE TO TYPE III PNEUMOCOCCUS—Clinically, these cases are characterized by an insidious onset with very little, if any, rise in temperature. The gross symptoms appear rather slowly and it is not until after several weeks have elapsed that meningeal symptoms or septicemia make their appearance rather abruptly and rapidly. The general symptoms consist of low grade toxemia, indisposition, and, occasionally, nocturnal pain. In many cases there are no warning signs until meningeal and septicemic symptoms appear.

On examination the drum membrane may be red and bulging when the disease is in the acute stage but as it passes into the latent stage there is a complete loss of the normal landmarks. One notes an occasional fulness in the posterior quadrant with the appearance of rugae. The discharge is scanty and mucoid. There is conspicuous absence of pain or tenderness over the mastoid. However, one may elicit pain on deep palpation. On percussion one may also note scalp tenderness over the temporal region. There is also conductive deafness during the early stages and as the infection progresses the perceptive element manifests itself.

Collateral Aid to Diagnosis of Acute Mastoiditis.—**Blood Counts**—The leukocyte count may

be used as a guide. When the total leukocyte count is on the decline, especially below 14 000, cells per cu mm of blood, and the polymorphonuclear count is still high, we may assume, at least from a clinical and laboratory standpoint, that the bactericidal power in the blood is at its highest point. Some authorities call this process the opsonic index. Whatever the underlying principles may be, complete blood counts should be performed at various intervals in order to determine the status of the hematopoietic system.



Fig 244 —Roentgenogram showing an acute coalescent mastoiditis

Roentgenologic Study—Comparative study between the normal and diseased mastoid should be made. The type of mastoid, whether pneumatic, diploic or sclerotic, should be noted (see Fig 220). The topographical and regional anatomy of the mastoid, its cellular distribution, the type of cells position of the sinus and its surrounding area should also be observed.

Roentgenograms are indispensable as an aid to diagnosis in cases of atypical mastoiditis in those of chronic mastoiditis with cholesteatoma, and in children with mastoiditis. In the latter when the cells are not fully developed, a roentgenologic diagnosis may often be made after the antrum has been located, as a few cells are invariably present there.

In an acute mastoiditis of recent origin there is first hyperemia, and the congestion is the same as in any other part of the body. Roentgenologic studies of both mastoids reveal that the normal side is clear and that the cellular

components stand out sharply with distinct cellular septa, while in the congested side all the normal elements of the mastoid are present, but in addition there is a slight haziness. During this stage either resolution takes place or the pathologic process continues. In the latter instance it may be assumed that the bacteria are active and that the mucous membranes lining the mastoid cells are becoming more edematous. There is also a constant infiltration of round cells and an increase in the leukocytes, and the congested area gradually becomes purulent. The roentgen ray plate at this time will reveal marked cloudiness with a tendency to obliteration of the cellular spaces which appear as though they were filled almost to the brim

with an extension of the haziness towards the squamous portion of the temporal bone is suggestive of an epidural abscess.

Treatment of Acute Mastoiditis—*Abortive Treatment*—An ice bag or heat should be applied locally, heat is preferable. Sedatives are indicated since the pain is worse at night and in the morning.

Sulfonamide therapy should be instituted promptly, the dosage being 1 grain (0.065 gm) per pound of body weight for the first twenty-four hours. Sustained therapy should be continued every four hours. The blood sulfonamide concentration should not exceed 10 mg per 100 cc. The patient must be kept under close supervision as the drug may mask symptoms of a



Fig. 245—Left view illustrates a normal pneumatized mastoid process. Right view illustrates a cholesteatoma.

The cellular septa are still visible as a minute network of fibrillated strands and the appearance of the plates at this stage indicates that exudation is present. With the advent of an increase in the purulent constituency there is also an increase in the intercellular and intracellular pressure. The picture now presents a distinct opacity with a blurring of the cellular septa. Absorption, liquefaction and cavity formation appear on the roentgen ray plate as a dark spot or as a shadow more or less regularly outlined, with the edges blurring and no trabecula visible. In contradistinction, a normal large cell is irregular with the edges sharply outlined, and the cellular septa are distinctly visible. A loss of cellular structures over the zygomatic area

spreading lesion. The blood concentration should be maintained until signs of mastoid involvement have disappeared and then the dose should be reduced by one half and the therapy continued for several days. If the infection progresses surgical intervention may become necessary.

Plenty of fluids, fruit juices, and alkalis should be prescribed to prevent acidosis. Cathartics are indicated to clear the gastrointestinal tract. Tonics and blood transfusions are desirable. Heliotherapy (infra red and ultraviolet) may be very helpful. Roentgen ray therapy (75 R to 100 R for several days) may be beneficial.

Local treatment consists in mopping the ex-

ternal auditory canal, irrigating the ear, and instilling sulfathiazole or sulfanilamide drops (2 per cent solution), or neutral acriflavin (2 per cent solution) into the canal. The irrigation may be accomplished by the dropper, rubber-bulb or fountain syringe method. A normal saline, boric acid, metaphen (1:10,000), bi-chloride of mercury (1:20,000), or cresalol compound (1 teaspoonful to 1 quart of water) solution may be used. The solution should be heated to 110° F. Under local treatment, the external auditory canal should be protected from abrasions by application of an ointment such as 1 per cent yellow oxide of mercury or 5 per cent sulfathiazole ointment.

Operative Treatment—This consists in a simple mastoidectomy performed either by the postauricular or endaural routes.

TECHNIC OF SIMPLE MASTOIDECTOMY (POST-AURICULAR ROUTE)—A postauricular incision is made through the soft parts and the external surface of the mastoid process, *i.e.*, the field of the operation, is exposed. The skin and periosteum are elevated and the mastoid cortex is exposed. With scissors, part of the attachment of the sternocleidomastoid muscle is removed.

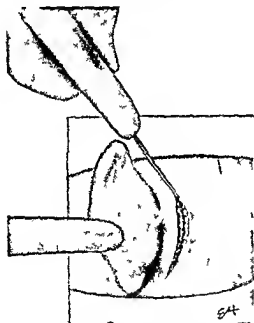


Fig 246—Postauricular incision for simple and radical mastoidectomy

The mastoid process, *i.e.*, (1) the linea temporalis or posterior ridge of the zygoma, (2) the spine of Henle, and (3) the mastoid tip, is visualized. The cortex is removed with a gouge, mallet, or dental drill. The mastoid cavity is

exenterated with a curet, all necrotic bone, diploic bone, granulation tissue, polyps, pus, et cetera being removed. The lateral sinus plate, the dural plates, Trautmann's triangle, Citelli's angle (sinodural angle), the horizontal semicircular canals, Richard's solid angle, the vertical portion of facial canal, the perilymphatic cells, et cetera are visualized. The zygoma

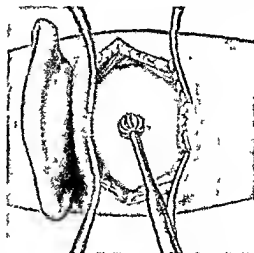


Fig 247—Exposure of mastoid cortex

is excavated anteriorly, the subarcuate region is skeletonized, and the antrum is exposed. When the sinus or dural plates are necrosed, the diseased portion is removed so as to facilitate drainage and to permit visualization of the underlying structures. The wound is irrigated with normal saline solution. A drain (*i.e.*, rubber tube, rubber dam, or plain or iodoform gauze) is inserted into the antrum. The wound may be allowed to fill with blood to form a blood clot dressing. The wound may be filled with sulfanilamide crystals before closing it by means of silk, wire, clips, or dermal sutures. A primary or complete closure, depending upon the surgical judgment, is made. The external auditory canal is packed with $\frac{1}{4}$ - to $\frac{1}{2}$ -inch gauze in order to prevent atresia of the external auditory canal.

The dressing should not be removed for seventy-two hours postoperatively unless there is a rise in temperature or there is evidence of toxic symptoms. At the first dressing the packing is removed from the external auditory canal. Should the middle ear be dry, the postauricular drain is removed. Politization and catheterization are instituted at this time to ventilate the middle ear and thus prevent adhesions and deafness. On the fifth postoperative

day the sutures are removed and the patient is permitted to be out of bed if the temperature is normal

thetic is to be employed, 1 per cent novocaine in adrenalin (1:15,000) is injected in the operative area. The membranous part of the external

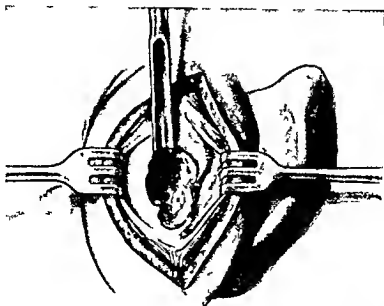


Fig. 248—Removing the cortex of the bone by means of the chisel (From Levine: Practical Otology, Lea & Febiger)

ENDAURAL APPROACH (LEMPERT TECHNIC)—When general anesthetic is employed a few drops of a 1:15,000 solution of epinephrine are

auditory canal is stretched and widened with a nasal speculum. The *first incision* is made in the superoposterior wall at the junction of the

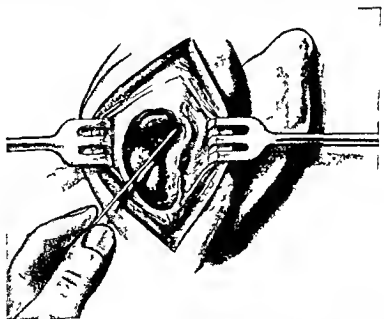


Fig. 249—Locating the mastoid antrum with the feeler probe (From Levine: Practical Otology, Lea & Febiger)

injected into the skin and periosteum of the membranous portion of the posterior external auditory canal wall at a point internal to the anterior border of the concha. If a local anes-

thesic is to be employed, 1 per cent novocaine in adrenalin (1:15,000) is injected in the operative area. The membranous part of the external

canal wall until the lower end of the anterior border of the concha is reached. A *second incision* is made in the superoposterior wall of the external auditory canal wall at the point of commencement of the first incision and continued outward along the superoposterior canal wall up to the antauricular suprameatal membranous triangle. The speculum is discarded and the auricle pulled upward with the thumb and index finger, thus bringing the surgical triangle

rest of the membranous layers down to the bony surface of the temporal bone. The periosteal elevator is inserted into the first incision and while the underlying surface is hugged with the edge of the instrument the entire triangular flap is lifted subperiosteally from its bony surface. The flap is now grasped with a hemostat and is completely removed with the aid of curved scissors. By the complete removal of this membranous flap an endaural extracartilaginous window is created for an approach to the mastoid process of the temporal bone. Exposure is maintained either by a self retaining retractor or by two four pronged retractors held by an assistant. Once the mastoid cortex is exposed, a dental drill, gouges, mallet, and other instruments may be employed. The operation is then continued following the technic of simple mastoidectomy.

POSSIBLE DANGERS IN MASTOIDECTOMY — These consist in (1) injury to the facial nerve, (2) penetration of the lateral sinus, (3) penetration of the dura, with injury to brain substance, implanting the infection, and inducing meningitis, (4) injury of the ossicles in the middle ear while probing the antrum, and (5) dislodging the incus, with resultant deafness.

TERMINATION — Resolution usually takes place. However, the disease may progress and one or more of the following conditions result: chronic suppurative otitis media, petrositis, osteomyelitis, pachymeningitis, diffused meningitis, septicemia, sinus thrombosis, labyrinthitis, facial palsy, impairment in hearing, deafness.

CHRONIC MASTOIDITIS

Chronic suppurative otitis media denotes a tympanic inflammation with a perforation of the drum head accompanied by a persistent flow of pus, with no tendency toward resolution. Chronic suppurative mastoiditis not only denotes an infection of the middle ear with involvement of the mastoid process but also signifies infection in other portions of the temporal bone.

Factors bringing about chronic suppuration of the temporal bone may be (1) A patulous eustachian tube which may become infected following swimming, exanthemas, sinusitis, rhinopharyngitis, tonsillitis, et cetera. (2) An infection of the eustachian tube which may spread by reason of the pus burrowing under the submucosa. (3) The presence of embryonic tissue. (4) The constant formation of fibrocon-

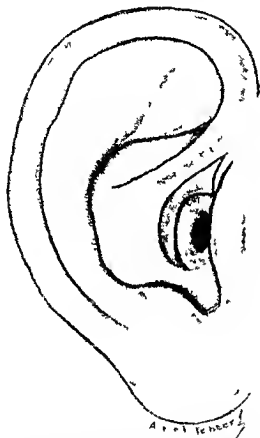


Fig. 250 — The creation of a mobile endaural membranous and extracartilaginous window for the antauricular surgical approach to the temporal bone showing three endaural incisions. (From Lempert Arch Otolaryng vol 28)

into view. The second incision is then continued into and through the anterior border of the antauricular suprameatal triangle, adjacent to the tragus up to the apex of this triangle where the helix and tragus nearly meet. A *third incision* is made to connect the outer ends of the first two incisions. The anterior border of the concha is brought into prominence by pulling the auricle backward. The incisions should be carefully outlined through the skin only. Then they are carried deeper through the

nective tissue in the middle ear with retention of secretions (5) Formation of granulation tissue and polyps (6) Infection of the middle ear involving the epitympanic region, attic, and aditus ad antrum (7) Ingrowth of squamous epithelium through a marginal perforation in the drum head (8) An acute inflammatory process in the mastoid which may have failed to undergo resolution (9) A chronic petrositis (10) Osteomyelitis of the temporal bone (11)

temporal bone, namely, the benign type and the perilous type.

Benign Type—In this type the structures involved are the mucous membranes of the eustachian tube and of the middle ear, part of the drum membrane, the attic, and, occasionally, the aditus ad antrum. Among the symptoms the discharge is the most important. Suppuration may be thick or thin, copious or scanty, mucoid (as it occurs in eustachian disease) or mucopur-

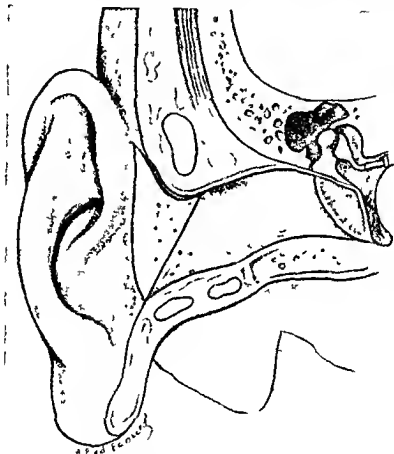


Fig. 251—The creation of a mobile endaural membranous and extracartilaginous window for the antauricular surgical approach to the temporal bone showing section view of the three endaural incisions. (From Lempert Arch. of Otolaryng. vol. 28)

Exanthemas, particularly acute necrotic otitis media occurring after scarlet fever, and leading to chronic suppurative otitis media.

The bacterial flora may include (1) *Streptococcus haemolyticus*, (2) *Streptococcus non-haemolyticus*, (3) pneumococci, (4) *Bacillus coli communis*, (5) Friedlander's bacillus, (6) bacillus of Vincent's angina, and (7) *aspergilli*.

Types of Chronic Mastoiditis.—There are two clinical entities in chronic suppurations of the

ulent, which usually is found in conjunction with mixed infections. It is generally odorless, there being no bony necrosis.

On otoscopic examination there may be seen a large central perforation without apparent destruction of the ossicles. On the other hand, a marginal perforation may be found. In such cases, however, no ingrowth of epithelium is seen. A perforation in the anterior inferior quadrant may also be noted. This is usually significant of eustachian involvement. Bony

necrosis, granulation tissue, or polyps are seldom found in the benign type of chronic mastoiditis.

There is a gradation of impairment in hearing in every case of suppurative otitis media. The factors bringing about the hearing loss may be adhesions, fixation and ankylosis of the stapes, changes about the oval and round windows, destruction of the tensor tympani and stapedius muscles, and/or partial or complete loss of the tympanic membrane. The hearing loss is of the

lining of the middle ear, and part of the drum membrane. In addition, there may be an involvement of the ossicles, the aditus ad antrum, Prussak's space, and any part of the temporal region which may include the labyrinth, the dural plate, meninges, facial canal and nerve, and the lateral sinus.

The type and character of the suppuration is of utmost importance in establishing the diagnosis. Suppuration differs in each case. For example a profuse, thick, and creamy discharge is

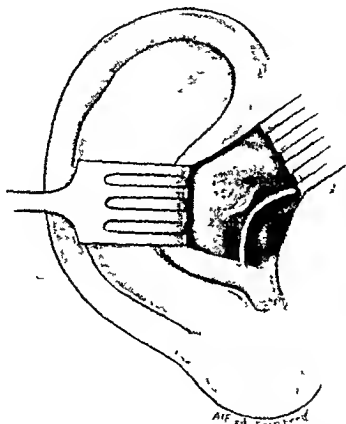


Fig 252—The creation of a mobile endaural membranous and extracartilaginous window for the antauregular surgical approach to the temporal bone, showing completion of the antauregular exposure of the mastoid process (From Lempert Arch of Otolaryng vol 28)

conductive type and consists of the loss of the lower tone limits with a diminution in air conduction. The vestibular examination generally shows normal reactions. However, whenever there is a large perforation, the caloric reaction produces a much more rapid response on the affected side because of the direct contact of the fluid with the vestibule.

Perilous Type.—In the perilous type of chronic mastoiditis the structures involved are the eustachian tube, the mucous membrane

characteristic of mucous membrane destruction. A profuse, caseous, and offensive discharge is characteristic of bony necrosis. A thick and extremely offensive discharge very often is characteristic of cholesteatomatous masses in the attic. The odor may also be attributed to decomposition of the squamous epithelium and pus, or to bony necrosis resulting from the pressure of the cholesteatomatous mass.

On otoscopic examination one or more perforations, granulation tissue, and large or small

polyps may be seen. The location of the perforations is of diagnostic importance. For example, a perforation along the floor of the drum head is often significant of necrosis of the roof of the jugular bulb. A perforation along the antero-inferior portion of the drum indicates involvement of the eustachian tube perforations in Prussak's space, necrosis of the tympanic ring or disease in the attic, aditus ad antrum, a perforation in the superior margin

encountered when cholesteatomatous masses are present and may be attributed to the in growth of the squamous epithelium.

Granulation tissue and large or small aural polyps may be found anywhere in the middle ear and signify bony necrosis. One may see large polyps which occasionally extend outward into the external auditory canal. One may also note small polyps which seem to grow out from the edges of the drum head from the middle

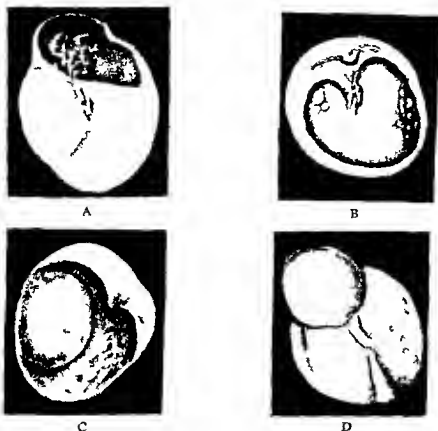


Fig. 253—A Large perforation in Shrapnell's membrane exposing a carious ossicle. B Large perforation of the drum membrane exposing the remains of a carious malleus. Inflammatory granulations are visible in the tympanic cavity. C, Large perforation of the drum membrane with an absence of the handle of the malleus. A polypoid excrescence is seen protruding from the tympanic cavity into the external auditory canal. D Aural polyp protruding from a small perforation on the upper portion of the drum membrane into the external auditory canal. (From Oppenheimer, *The Surgical Treatment of Chronic Suppuration of the Middle Ear and Mastoid*, The Blakiston Company.)

is also indicative of some involvement in the epitympanum. Perforations along the posterior wall of the drum may be due to bony necrosis of the mastoid or to necrosis of the adjacent bony structures. A perforation at the umbo may be due to necrosis of the handle of the malleus. Perforations anywhere in the drum head may result from necrosis of the ossicular chain or other factors. A large marginal perforation accompanied by an offensive discharge is often

ear or from the necrotic ossicles. At times polyps spring from such vital structures as the dura, lateral sinus and labyrinth. Polyps and granulation tissue result from either bony necrosis or the overgrowth of fibroconnective tissue with an attempt to heal. (See Fig. 255.)

Conductive and perceptive deafness are characteristic findings. Where there is complete deafness there is usually a history of labyrinthine symptoms. Tinnitus is present in some

cases although not a constant symptom. Vertigo and nystagmus are not common findings and may be due to toxicity, irritation of the labyrinth, or erosions in the petrous portion of the temporal bone.

The gross general symptomatology of a complicated chronic suppurative ear depends upon

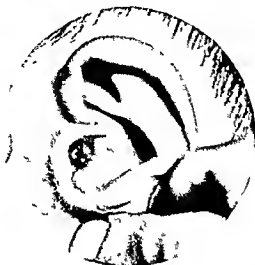
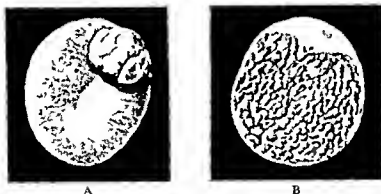


Fig. 254—Aural polyp

the structures involved, namely, the labyrinth, the lateral sinus, the cerebral and cerebellar dura, the facial nerve, and the meninges. If the labyrinth is involved, the patient has vertigo, dizziness and nystagmus, and the fistula tests

a positive Toby-Ayer test. Other symptoms likely to be present are papilitis, choked disk, and positive Griesinger's and Crowe-Beck signs. If the cerebral and cerebellar dura are involved, there will be symptoms of meningitis and circumscribed pachymeningitis which may become manifest in the form of pain over the occipital and temporal regions, severe and unilateral headache over the same areas, and, occasionally, projectile vomiting.

The neurologic symptoms are governed by the extent of involvement. If the meninges are involved, whether by a circumscribed or a diffuse purulent leptomenigitis, motor and sensory disturbances will occur. The sensory disturbances reveal themselves in anesthesia, paresthesia, hyperesthesia, and hypoesthesia. The motor disturbances are stigmatized by paresis or palsies, or by hyperactive reflexes and convulsions. Sustained temperature, headache, outcries, irrationality and stupor, vomiting and nuchal rigidity, and other signs indicate active meningitis. This diagnosis, however, should be verified by spinal puncture and study of the spinal fluid. If the facial nerve is affected, there will be a loss of taste, widening of the palpebral fissure, and inability to frown or to move the lower lid upward. The patient will have a smooth face on the side involved, because of the loss of innervation of the muscles of expression.



A

B

Fig. 255—A, Aural polyp protruding through Shrapnell's membrane. B, Polypoid granulations. (From author's article in Nelson's Loose Leaf Surgery of the Ear [edited by Kopetzky], Thomas Nelson & Sons.)

will be positive. If purulent labyrinthitis is present, there will be an elevation of temperature and other toxic symptoms. If the lateral sinus is involved, there will be a septic temperature, which may be remittent or intermittent, accompanied by chills, occasional convulsions, a pathologic blood picture in approximately 50 per cent of the cases, embolic phenomena, and

The possible complications of the perilous type of chronic mastoiditis include suppurative labyrinthitis, cerebellar abscess, cerebral abscess, meningitis, and infection of the sigmoid (lateral) sinus.

Treatment of Chronic Mastoiditis.—Before suggesting rational lines of treatment one must classify the particular case in question.

Pus is attacked by one of the following means (1) In the moist method, the pus is broken up by means of irrigation with hydrogen peroxide or sodium bicarbonate solution, the odor is eliminated by potassium permanganate irrigations, and irrigations with solutions of phenol, saline, boric acid, or metaphen (1:5000) are given (2) In the dry method, the canal is mopped with cotton until dry, the ear is insufflated with iodine or boric acid powders, and some of the following solution is instilled

Formalin	5 minims
Ethyl alcohol	1 drachm
Distilled water	sufficient quantity to make 1 ounce

A prescription for the solution may be given and the patient instructed to place 5 drops into the canal allowing it to remain from five to ten minutes before draining and mopping the canal. This procedure should be repeated three times a day.

Ionization (zinc sulfate, $\frac{1}{2}$ per cent) therapy may be utilized.

Granulation tissue or aural polyps may be attacked by means of (1) Chemical caustics, such as chromic acid or trichloroacetic acid applications, or silver nitrate bead (2) Cauterization (3) Removal of the growth by destruction of its root or by removal of the cause (Polyps may be removed through the external auditory canal, either whole or in part, by means of the snare or sharp ring curet. If the polyps spring from the inner tympanic wall, and if there is a tendency for rapid reformation of the growth there is sufficient reason to perform a radical operation.) (4) Conservative surgery consisting of ossiculectomy (5) Intratympanic attic surgery (6) Tympanomastoidectomy (radical mastoidectomy) either by the endaural or postauricular route.

Technic of Tympanomastoidectomy (Radical Mastoidectomy — Postauricular Route)—The technic of a simple mastoidectomy (postauricular route) is followed. The mastoid cells are completely exenterated. The posterior canal wall is removed, thus converting the middle ear and mastoid antrum into one cavity. The subarcuate region is skeletonized. The structures in the middle ear, *i e.*, drum, malleus, and incus, are removed. The stapes must not be disturbed as it closes the oval window leading into the labyrinth. Polyps, granulation tissue, pus, and cholesteatoma are removed from the middle ear

using Hartmann's forceps and curet. The hypotympanic region is curetted, also the epitympanic region. The eustachian tube is exposed. The peritubal cells are exenterated. The cochleariform process and the tensor tympani muscle are removed. The following structures are visualized: the promontory, the cochlea, the horizontal semicircular canals, Richard's solid angle, the dorsal plate, the sinodural angle, Trautmann's triangle, the hypotympanic region, the epitympanic region, the eustachian tube, the posterior surface of the temporomandibular joint, and the facial canal (horizontal and vertical portions). The mastoid wound is closed and drainage is established through the external auditory canal. Healing may be promoted by a plastic flap, utilizing the derma from the external auditory canal by the Thiersch skin graft.

NOTE. One of the pitfalls in mastoid surgery is injury to the facial nerve. In a simple mastoidectomy one may injure the vertical portion of the facial nerve. In the radical or tympanomastoidectomy, the horizontal and the vertical portions of the facial nerve may be injured. In simple mastoidectomy, the horizontal semicircular canal is the great guide in avoiding this pitfall. One must therefore exercise extreme caution when operating below that depth. In radical mastoidectomy, after removal of the granulation tissue and polyps with Hartmann's forceps, one must be extremely careful in exposing the horizontal portion of the facial canal. Once this portion is exposed the danger of injury to the facial nerve is averted.

Sulfanilamide powder in the mastoid cavity has a tendency to cake and create pressure on the facial nerve with resultant facial weakness and paralysis. Its use is therefore contraindicated when the facial nerve is exposed. In several cases observed by the author such paralysis appeared on or about the fifth day following the operation, and necessitated the removal of the encrusted powder from the surgical field to relieve the pressure.

SINUS PHLEBITIS

In the parlance of the otologist, phlebitis and lateral sinus thrombosis are synonymous. Both terms denote inflammation of the vein involving the intima, media, and adventitia.

Phlebitis and lateral sinus thrombosis may result from (1) irritants, which may be chemical or bacterial or mechanical, such as an injury during surgery, (2) the extension of infection from the middle ear or mastoid by continuity or contiguity, (3) chronic suppurative otitis media and chronic mastoiditis with direct involvement of the vessel, (4) bacteremia, or (5) hemolytic streptococcal infection, which is most prevalent. They may begin because of changes

in the vessel wall affecting the intima, media, and adventitia, changes in the vasovesorum interfering with the nourishment of the vessel, or changes in the blood elements and local circulation. The three underlying principles in the formation of a thrombus include slowing of the circulation, injury or changes in the endothelium of the intima, and organisms or toxins. If only the vein is involved, there is a phlebitis, if

Symptoms—Sinus phlebitis is manifest by severe headache, which may be general or localized at the site of the sinus. There is a high fever, septic in character, with remissions and intermissions (see Fig 256). The pulse rate is in proportion to temperature (A rapid or feeble pulse is significant of myocardia, or pre-mortal state). Violent chills and rigors are noted. Respirations are increased. Great pros-

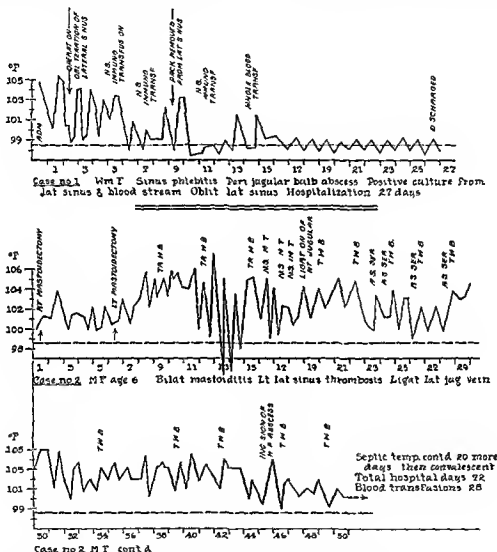


Fig 256—The above chart clearly illustrates a typical temperature graph in phlebitis, thrombosis, and septicemia.

the vein and the blood elements participate (*i.e.*, fibrin, blood platelets, red blood cells, white blood cells), a thrombus results. The thrombus may be either parietal or mural, sterile or infected. It may spread in the direction of the circulation or extend proximally or distally. The central portion is ordinarily infected, while the distal and proximal ends are frequently sterile.

tration is evident in many cases, particularly when the temperature is high and when there is profuse sweating. The mentality is unaffected except during extreme hyperthermia. Cyanosis may be noted. Convulsions occur in children. Nausea and vomiting are frequently observed. Diarrhea is present in occasional cases. Optic neuritis or choked disk may be noted.

When the lateral sinus is thrombosed pain

and tenderness are present along the course of the sternocleidomastoid muscle also pain and tenderness to palpation along the jugular vein which feels cord like if the vein is thrombosed. Occasionally there is edema of the face, occipital region, and posterior triangle of the neck (the facial edema being due to faulty collateral circulation). Edema of structures in and about the mastoid on the side of the sinus involved is due to a thrombosis of the condyloid veins (This formulates Griesinger's sign). Occasional flushing or engorgement of the conjunctival vessels is the result of partial obstruction of the jugular vein. The spinal puncture reveals increased pressure. Pressure on the jugular vein on the side of the lateral sinus involved fails to give a rise in manometric reading of cerebrospinal fluid pressure (This is designated as the positive Tobey Ayer Queckenstedt sign).

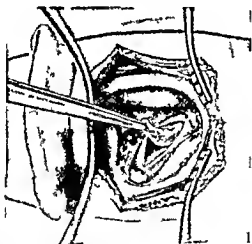


Fig. 257—Packing of sinus incision on the vessel and removal of the clot

Involvement of the lateral sinus with obstruction serves to embarrass intracranial circulation until collateral circulation is established. This is owing to the close interdependence between the venous and arterial supply of the brain. The rise in cerebrospinal fluid pressure is manifested in the form of meningeal symptoms: (1) the fundus shows a papillitis and choked disk; (2) convulsions as noted in children are due to increased cerebrospinal fluid pressure; (3) Kernig's and Brudzinski's signs are positive; and (4) embolic phenomena are noted.

Study of the blood shows the hemoglobin content and red blood cells to be reduced. Leukopenia exists whenever there is an over-

whelming infection. There is a leukocytosis ranging from 20 000 to 25 000 cells per cu. mm and a degeneration of the mature leukocytes. The Stilling index reveals a shift to the left. The blood culture is positive in 40 per cent of the cases.

NOTE. It is best to take the blood at the maximum height of the temperature and to use both aerobic and anaerobic culture media. With these precautions one may get a higher percentage of positive cultures.

Medical Treatment.—This includes the administration of hemotonics and stimulants. Chemotherapy, one of the sulfonamides or penicillin should be instituted.

NOTE (1) Sulfonamide blood concentration should be at least from 10 to 15 mg. per 100 cc. of blood. (2) Before the employment of penicillin it is of utmost importance to determine what organism is responsible. All other therapy must then be stopped. When penicillin is given by repeated intravenous injection in infections caused by hemolytic streptococci, staphylococci or pneumococci 15 000 to 20 000 units of penicillin should be given initially followed by 5000 units every hour. If constant intravenous injection is used 5000 to 10 000 units may be administered per hour. After the temperature has returned to normal, the total dose may be reduced by one-half and this amount continued for one or two days. As this drug is excreted very rapidly it becomes necessary to repeat the dosage frequently.

Sera may be given. The patient should have daily blood transfusions of 1 to 1½ cc. per pound of weight. The transfusions may be direct or citrated blood may be used. Nonspecific immune transfusions are beneficial. Hypertonic solutions are given when the blood pressure and pulse warrant their administration. Heparin is given to prevent thrombosis.

Surgical Treatment.—A simple mastoidectomy or a revision of the operation is performed. The lateral sinus is exposed by removing the sinus plate. The sigmoid sinus is palpated for patency fluctuation or pitting. A needle is inserted into the vessel and the contents of the vessel are aspirated. The sinus is packed above and below with iodoform gauze in order to control bleeding. The vessel is incised and the clot removed (see Fig. 257).

The orthodox routine procedure is to establish patency and bleeding of the sinus at the proximal and distal ends and then ligate the internal and jugular veins. This may be accomplished by suction curet or forceps. The lateral sinus is repacked with plain or iodoform gauze.

in order to create a new thrombus and to control bleeding. It is the author's method to perform a thrombectomy *without* jugular ligation, as it has been found that the collateral circulation is so great that ligation does not prevent metastasis or embolic phenomena. In the event that the internal jugular vein is involved it may become necessary to ligate, excise, or to perform a phlebectomy in order to promote drainage.

INSTRUMENTS, DRUGS, AND SUPPLIES FOR SIMPLE MASTOIDECTOMY, TYMPANOMASTOIDECTOMY (RADICAL MASTOIDECTOMY), AND ENDAURAL APPROACH OF MASTOIDECTOMY (LEMPERT TECHNIC)

NOTE. No two surgeons use identical instruments for the performance of any given operation. Every surgeon has his favorite instruments which are best suited to his individual needs. The following suggestions for the operative layout include the basic instruments required for the performance of a mastoidectomy.

1. Knives or scalpels (2)—one for skin incision, one narrow long blade for plastic flap.

POSTOPERATIVE TREATMENT

<i>Acute Mastoiditis</i> (Simple Mastoidectomy)	<i>Chronic Mastoiditis</i> (Radical or Tympanomastoidectomy)	<i>Sinus Phlebitis and Lateral Sinus Thrombosis</i> (Thrombectomy and/or Internal Jugular Ligation and Obliteration of the Lateral Sinus)
1 Sedation 2 Gastro-intestinal care 3 Alkalinization 4 Fluids 5 Full diet after 24 hours if temperature is normal 6 Chemotherapy optional	Sedation Gastro-intestinal care Alkalinization Fluids Full diet after 24 hours if temperature is normal Chemotherapy optional	Sedation Gastro-intestinal care Alkalinization Fluids Semisolid diet.
7 Blood transfusions optional	Blood transfusions optional	Biochemotherapy essential as long as there is a positive culture (employ either a sulfonamide or penicillin). When penicillin itself has proven to be ineffective, then a combination of the sulfonamides and other supportive therapy should be supplemented.
8 First dressing after 72 hours—then remove packing	First dressing after 72 hours. Remove packing on the 6th day.	When sulfonamide therapy is employed blood transfusion is essential, when penicillin is employed no other therapy should be used in order that one may evaluate the efficiency of the drug. Remove packing at the end of the 7th day postoperatively when the lateral sinus has been obliterated.
9 Politzerization (only when ear is dry)	No politzerization ✓	No politzerization
10 Heliotherapy	Heliotherapy	Heliotherapy
11 Vitamins (thiamine hydrochloride, nicotinic acid, et cetera)	Vitamins, (thiamine hydrochloride, nicotinic acid, et cetera)	Vitamins (thiamine hydrochloride, nicotinic acid et cetera)

Prognosis.—The prognosis is good under skilful medical and surgical management.

Complications.—Embolism with infarction involving the visceral structures, such as the lungs, kidneys, or liver, is a serious complication and often proves fatal. When emboli terminate extravascularly, as in the joints or muscles, there is usually a good recovery. Suppuration occurs if the emboli are infected.

2 Hemostats (10)

3 Periosteal elevators

4 Retractors (4)—two rake retractors, two Jansen (self retaining type)

5 Scissors (2)—one Mayo, one curved

6 Forceps—anatomic mouse toothed bayonet, angular and Hartmann's forceps to remove malleus and incus, one cup-shaped and one plain forceps

7 Chisels—one flat chisel, other types and length to suit surgeon

8 Gouges—#14, #12, #10, #8, #6, #4

9 Mallet—metal preferred

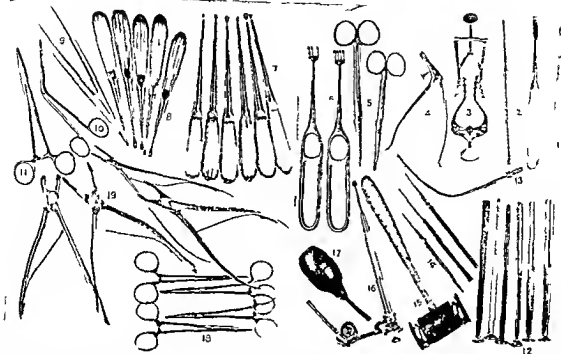


Fig 258—Instruments 1, Bard Parker knife, 2, periosteal elevators, 3, Jansen self retaining retractors, 4, aural nasal speculum for endaural operation 5, Mayo and curved scissors, 6, two rake retractors, 7, Lempert curets, 8, curets, 9 forceps—plain and tooth 10, Hartmann's forceps 11, needle holder 12, chisels, 13, suction tube, 14, probes, 15, mallet, 16, dental drill and chuck, 17, bulb syringe 18, hemostat, 19, rongeurs

10 Curets—one set of six different dimensions for endaural approach, Lempert long curets (sizes #4, #3, #2, #1, #00)

11 Rongeurs

12 Burs—motor and motor driven (Dental drill is optional, this requires a foot switch to operate motor) Mastoid burs must be sharp and of various sizes and shapes to fit handle

13 Probes—straight and curved

14 Needle holders

15 Needles—medium large, and curved

16 Catgut for ligation—#1 and #2 plain

17 Various sized rubber tube drains

18 Bone wax

19 Clips, clip holder, and clip remover

20 Razor, small wooden board and several needles where Thiersch skin graft is employed in radical mastoidectomy

21 Mastoid dressings

22 Drugs—alcohol, adrenalin solution (1:1000), neosynephrin for hemostasis in radical mastoidectomy

MATTHEW S ERSNER

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SUPPURATION OF THE PETROSAL PYRAMID

Acute Petrositis.—This condition, long known as a pathologic lesion, has only in recent times been given clinical recognition. Its features are a purulent lesion involving the cell and, sometimes, the diploic structures surrounding the cochlea, the semicircular canal, the internal auditory meatus, and the region of the temporal bone, described as the petrosal tip. It has been variously designated as *acute apicitis*, an *acute purulency of the petrosal pyramid*, *acute purulent osseous perabyrinthitis*, or *acute empyema of the petrosal tip*.

Etiology.—The exciting factor in this disease

is an infection of the tympanic cavity. The predisposing factor is an acute purulent otitis media accompanied by an acute mastoiditis.

Pathology.—The lesions fall into two gross types. One comprises the changes attending an osteomyelitis. This type usually occurs only in infants. The more common and usual type is a coalescent osteitis of the trabeculae of bone forming the intercellular walls of the pars petrosa. The lesion is produced by a suppuration which breaks down the intercellular walls in the bone, causing a coalescence and the localization of an empyema within the confines of the bone located in the pars petrosa. At the

an outlet under the skin in the upper triangle of the cervical region as a soft tissue abscess formation.

The lesion therefore, according to the time when it is observed, is either *intrapetrosal* or *extrapetrosal*. Where the lesion is located within the anatomical boundaries of the pars petrosa, it is termed intrapetrosal. When rupture has taken place, it becomes extrapetrosal. In each of these stages it presents specific symptoms which will be commented upon below.

Occasionally, the rupture of pus from the pars petrosa does not take place either endocranially or in the form of a "gravity abscess,"

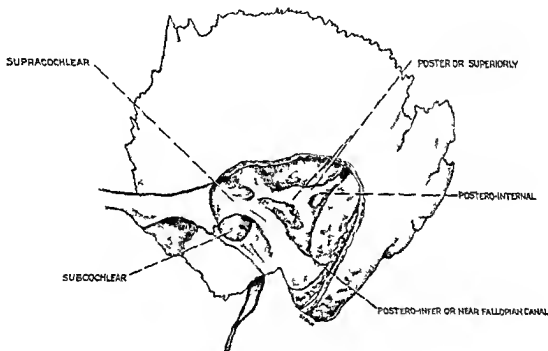


Fig. 259.—Locations of frequently found purulent tracts

onset of the disease, this localization is very definite, and when present, presents in many instances specific localizing symptoms. Later, in the development of the lesion, the coalescence spreads and becomes generalized throughout the pars petrosa. If unrelieved, the lesion finally ruptures into the endocranium, producing as the lesion advances, an epidural abscess, or it may break through downward, forming a "gravity abscess," burrowing its way through and along the layers of muscle working its way inward into the tissue of the neck to appear on the lateral pharyngeal wall as an abscess, or it may spread externally toward the surface of the neck through the various facial layers. It seeks

but discharges itself extrapetrosally, forming a fistulous tract which empties into the structures comprising the mastoid process. Since petrositis is a complication of acute purulent otitis media, in most instances, the mastoid process and the petrous pyramid are simultaneously involved, but the symptoms from the mastoid process predominate and overshadow the symptoms from the petrous pyramid, so that the mastoid symptoms center attention upon that lesion and the petrous lesion is usually discovered only after the mastoid extirpation has taken place and the symptoms attending the mastoiditis have abated.

The fistulous pockets may be found after

to make a *diagnosis of location*, because if this can be done, the located region can be reached without extensive surgical explorations in areas other than those affected. The diagnosis then consists in (1) the establishment of the fact that acute petrositis is present and, (2) the determination of the location of the lesion within the petrous pyramid. In the majority of cases, a diagnosis of location is possible.

When the symptoms outlined are present, a roentgenologic examination of the base of the skull is made. Involvement of the petrous pyramid is indicated when the structure of the pyramid on the affected side differs from that of the opposite side. If a base plate film has been exposed at the time such patient was first observed, before the onset of symptoms from the petrositis, as is my custom, then comparison with that plate, and the plate taken when symptoms are present, gives further substantiating data.

The diagnosis of location depends upon a very detailed study of the symptoms which are presented clinically. The diagnosis of acute purulent petrositis of the coalescent type—the form of petrositis most common among adults—rests upon the finding of an increase in the purulent discharge from the middle ear after a mastoidectomy has been performed and the presence or absence of abnormality in the pyramid structure as noted on roentgenograms. These symptoms and pyramid defect denote coalescence of the pars petrosa.

If, on inspecting the wound in the mastoid process, very little discharge is found in the periantral region and much discharge is seen coming from the middle ear, the presumption is that the discharge originates in the antecochlear region. On the other hand, if the middle ear discharge is not increased or is absent, but the antral area shows considerable discharge with some granulation appearing around the region of the semicircular canals at the base of the pyramid, the presumption is increased that one is dealing with a postcochlear localization. Postcochlear areas can be reached through the simple mastoid cavity without disturbing the tympanic cavity. Antecochlear regions usually cannot be reached through the wound made in the simple mastoidectomy operation, but must be reached through the tympanic cavity.

No diagnosis should ever rest solely on roentgen ray examination, but if an involvement of the pars petrosa is shown on roentgenograms and symptoms of petrositis are present, the

diagnosis may then be considered as established.

DIFFERENTIAL DIAGNOSIS—In its early stage, irritation of the labyrinth, from any cause whatsoever, produces a vestibular nystagmus. Purulent osseous perilabyrinthitis also causes a nystagmus. If lateralization is towards the diseased ear, then the function of the cochlea is undisturbed and one may rule out acute labyrinthitis. Acute labyrinthitis is also ruled out if sound is heard in the cochlea of the affected side when the healthy ear is masked. On the other hand, if the cochlea does not perceive any sound and a vestibular reaction is present, while a roentgenologic examination reveals no abnormality, one may assume that one is dealing with a labyrinthitis (located in the perilabyrinthine area) rather than a suppurative petrositis located at the base of the petrosal pyramid.

Roentgenologic evidence of petrosal involvement in an ear that is not discharging and in which there is no indication of disease in the mastoid process, with symptoms which sometimes may point to involvement of the abducens, diplopia being present, may be indicative of a tumor of the petrosal pyramid, but does not indicate a suppurative petrositis.

Very often meningitis is already present when petrositis is suspected. High temperature, projectile vomiting, changes in the sensorium, increased cerebrospinal fluid pressure, and the appearance of pathologic cells in the spinal fluid as well as an increase in albumin and lactic acid and a decrease in the chloride and carbonate content with or without bacteria in the fluid, indicate that the lesion has reached the meninges and involves them. Petrositis may or may not be present as one of the intermediate steps of the meningeal involvement.

Treatment—Prophylactic treatment consists in the early recognition of involvement of the mastoid process and surgical intervention as early as indicated to relieve the specific symptoms. The very relief of the condition in the mastoid process may bring about resolution in the pars petrosa if it is infected. In older mastoid surgery a trephine opening into the mastoid process was found effective in stopping the development of further coalescence because of relief of the pressure which is partly at fault in producing a coalescent mastoiditis. A simple mastoidectomy also relieves this pressure and therefore further coalescence of the mastoid process ceases. If egress of the pus in the pars

simple mastoidectomy has been performed in the following order of frequency

1 Behind the semicircular canals emptying into the mastoid process in the region of Trautmann's triangle

2. Above the cochlea and discharging underneath the arch of the superior semicircular canal

3 It may discharge below the cochlea into the hypotympanum and the tympanic cavity

4 Lastly, from in front of the cochlea in the region of the orifice of the eustachian tube and the region where the carotid canal runs through the petrous portion of the temporal bone (See Fig 259)

Symptoms—The symptoms are general and local. An acute mastoiditis, which either preceded or is concomitant with the petrosal lesion, gives its own set of symptoms. When mastoidectomy has eliminated the symptoms which the infection in the mastoid process evoked, petrositis should be suspected as being present when there gradually develops retro-orbital, intra-orbital, or supra-orbital pain on the same side of the head to which the mastoid was found at operation to be diseased. The attacks of retro-orbital and supra-orbital pain are at first intermittent and usually begin nocturnally. The periods of quiescence gradually become lessened and the attacks of pain become more frequent. Often, but not always, a slight elevation of temperature is present. The temperature is never very high, not above 101° F. The differential blood count gives no determining data. The patient simply has these symptoms of pain from involvement of the first branch of the fifth nerve. Objectively, there is present distinct narrowing of the palpebral fissure on the side of the involved ear. Photophobia is occasionally observed.

If the acute involvement of the petrosal pyramid takes place after a mastoidectomy has been performed, and the middle ear has been free from discharge for an interval of time, simultaneously with the attacks of retro-orbital and supra-orbital pain, middle ear discharge reappears. When the discharge has not abated after mastoidectomy, the onset of the petrosal lesion may be signaled by an increase in the amount of middle ear discharge, and the reappearance, postoperatively, of the pulsating characteristics of the discharge from the middle ear.

As the lesion progresses the intervals between the attacks of pain gradually become shorter

until the pain is continuous and is marked by an increase in its intensity. A little later in the course of the disease, abducens palsy is observed and double vision is an added symptom. Occasionally patients present other transient symptoms. Thus, at times, a facial palsy is noted, which sometimes clears up within a few days. Also there are transient periods during which vestibular nystagmus is noted. This nystagmus is the result of the irritation caused by the development of the perilymphatic lesion in the region of one or other of the semicircular canals. As the disease progresses, in some instances there is a sudden decrease in the severity of the otorrhea, with the sudden appearance of a parapharyngeal abscess. This signifies the rupture, extrapetrosally, of the lesion into the lateral pharyngeal wall, and its drainage downwards through the abscess, resulting in the quiescent stage of symptoms. As one observes such a case, there suddenly is a cessation of all symptoms and the patient reports that he feels well. This *quiescent stage* is often the beginning of what has been termed "the asymptomatic stage of the lesion," and it may either signify the beginning of resolution or, in a lesion which progresses, a rupture into the endocranium. If the latter eventuates, this condition indicates the necessity of surgical intervention. The quiescent stage ends, after a variable interval of time, with the commencement of the *terminal stage* of the lesion which gives the symptoms first of invasion, and finally of infection of the meninges and the central nervous system, namely, the beginning of a purulent meningitis.

If a quiescent stage commences and the interval between the attacks of pain in and above the eye becomes of longer duration until there are days during which there is no recurrence of the pain, and if a decreasing amount of discharge from the middle ear accompanies these increasingly lengthening intervals of quiescence, then one can assume that the lesion is spontaneously resolving and that no surgical intervention is indicated.

It must be comprehended that in some cases, petrosal infection spontaneously resolves and heals, and in others, it progresses and requires surgical intervention. Once the lesion is diagnosed, surgical intervention should, as far as possible, be timed to eliminate the lesion before rupture has taken place extrapetrosally.

Diagnosis—In the diagnosis of acute petrositis in all instances an attempt should be made

to make a *diagnosis of location*, because if this can be done, the located region can be reached without extensive surgical explorations in areas other than those affected. The diagnosis then consists in (1) the establishment of the fact that acute petrositis is present and, (2) the determination of the location of the lesion within the petrous pyramid. In the majority of cases, a diagnosis of location is possible.

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Treatment—Prophylactic treatment consists in the early recognition of involvement of the mastoid process and surgical intervention as early as indicated to relieve the specific symptoms. The very relief of the condition in the mastoid process may bring about resolution in the pars petrosa if it is infected. In older mastoid surgery a trephine opening into the mastoid process was found effective in stopping the development of further coalescence because of relief of the pressure which is partly at fault in producing a coalescent mastoiditis. A simple mastoidectomy also relieves this pressure and therefore further coalescence of the mastoid process ceases. If egress of the pus in the pars

petrosa is afforded through any existing fistulous tract which empties into the surgical wound made in the mastoid process, the threat to life is lessened by that procedure, and further coalescence in the pars petrosa ceases, leaving only a suppurative tract which often heals after a long interval of time.

The treatment in all cases which are not on the road to resolution should be the minimum surgical intervention which is necessary to effect a cure. Routinized surgical procedure adopted to handle petrositis should be avoided since no

dedicated, a complete simple mastoidectomy is undertaken. If the roentgenologic examination revealed a suspicion of petrosal pyramid involvement and symptoms of such involvement are present, at the completion of the surgery on the bony parts a very careful search should be made for the purpose of locating purulent tracts and fistulas leading from the interior of the exenterated mastoid process into the pars petrosa. The search should start in the regions about the semicircular canals. When a tract is found, this tract should be carefully curetted

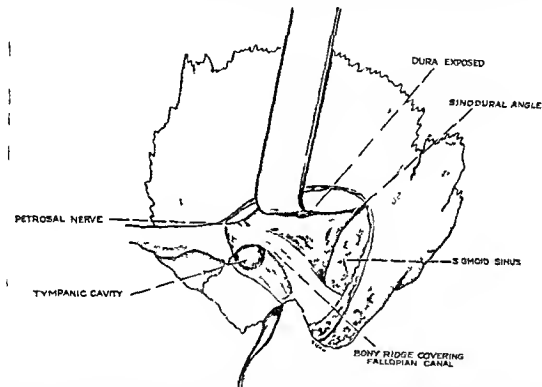


Fig. 260.—Schematic diagram showing a retractor elevating the dura of the middle fossa, the dura exposed, and the tegmen removed. The sigmoid sinus is exposed and the sinodural angle. A ridge of bone, including the bony posterior meatal wall and fallopian canal, is seen. More extensive removal of the squamous plate is often necessary.

one surgical procedure meets the requirements in all cases and in many cases much unnecessary surgery would result.

The decision as to the goal of surgical intervention is based upon an exact diagnosis of location. The location of the lesion having been determined, a procedure of surgical technique must be adopted to meet the situation and undertaken promptly.

EXTRAPETROSAL DRAINAGE—As soon as supuration occurs in the middle ear, drainage is established by myringotomy. If mastoid symptoms develop and surgical intervention is in-

toward the petrosal tip and drainage established, all removable diseased tissue being taken away with the aid of the curet. If no tracts are found and the purulency seems to be emanating mostly from the antecochlear area (the middle ear), then the posterior bony meatal wall must be broken down and the cavity converted into that of a radical mastoidectomy. After all the procedures of a radical mastoidectomy have been carried out a search is made in the antecochlear area for a fistulous tract leading into the petrosal pyramid. One may be found at the upper part near the tegmen or in the anterior

wall of the hypotympanum in the region of the orifice of the eustachian tube. This latter location is the most common.

Eagleton's Operation—Eagleton's operation is among those used and advocated by various authorities. Eagleton removes the tegmen cellulae, the tegmen antri and the tegmen tympani, performing a wide resection of the squama of the temporal bone. He carries his bony dissection outward and removes the base of the zygoma thus exposing the dura of the middle cranial fossa which he elevates carefully

into the meninges. The abscess is formed from a septic venous retrogressive thrombosis that finally breaks down to form an abscess in this area, which is found during operation without there being presented a rupture of the bone comprising the roof of the petrosal pyramid. Therefore the evacuation of the epidural abscess is not the objective of the Eagleton operation, but is incidental in entering the petrosal pyramid from above, and thus reaching the lesion which necessitated the surgery (See Fig. 260)

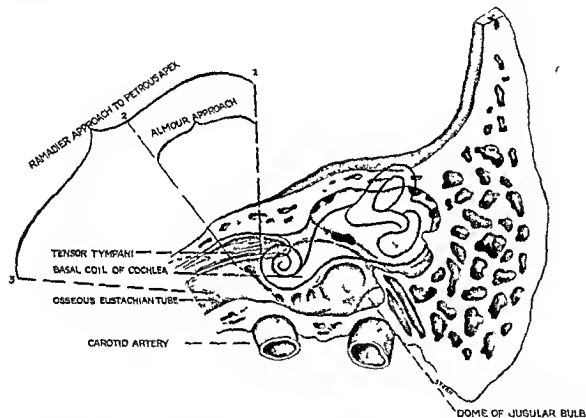


Fig. 261—Schematic diagram. The shaded portion denotes the triangular space the objective of the intra petrosal approach to the petrous apex. Area between 1 and 2 is the area of approach by Almour, area between 1, 2, and 3 approachable by Ramadier procedure.

from the bone upon which it lies, gradually working forward over the tip. By these procedures a coexisting extradural abscess from the rupture of a lesion in the petrosa is evacuated. An epidural abscess does not, however, always ensue from a rupture through the roof of the petrosa. I have even found it present with no break in the contiguity of the roof of the petrosa. In such instances, the extradural abscess located over the tip of the pars petrosa occurred from regression thrombosis of the skeletal veins which run through the petrosa

INTRAPETROSAL DRAINAGE—The Ramadier and the Almour Operation—Our efforts have been concerned mostly with better diagnosis and handling of the lesion while it is still intrapetrosal. Therefore, intrapetrosal drainages of the petrous apex, as devised by Almour and by Ramadier, come into consideration. In either Almour's or Ramadier's method a radical mastoidectomy is performed as a first step in the procedure. Entry into the petrosal tip is then made through a triangular area bounded in front by the carotid artery in its canal and in

the rear by the cochlea. The tegmen tympani forms the roof of this triangle (See Fig. 261.)

In the operation devised by Ramadier the tympanic plate of the external auditory canal posterior to the suture line at the base of the glenoid fossa is all removed and the internal carotid artery is located within its bony canal in its course through the temporal bone. The artery is pushed forward and through its bed which is thus exposed in the canal. Entry is made into the petrosa and, if apicectomy

auditory meatus. It is the only means by which collection of pus located in this region can be drained. It is also the only means by which, in the presence of a living, functioning membranous labyrinth, this area can be treated surgically and the hearing functions of the labyrinth be conserved. This area can be reached through the transverse labyrinthine route, but then all the structures of the labyrinth are destroyed in the process, with a resulting loss of function.

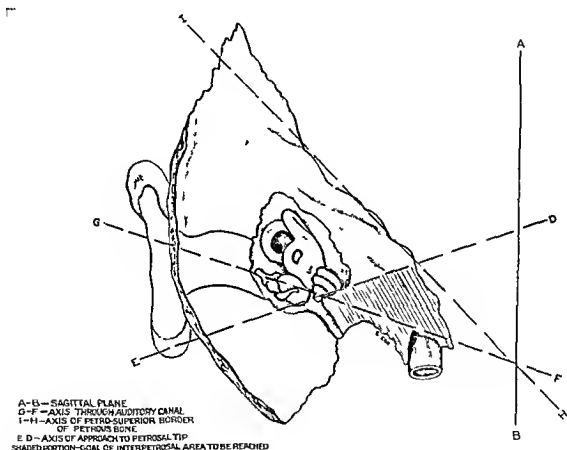


Fig. 262—Schematic diagram through the temporal bone

(evisceration of the apex of the petrous pyramid) is not deemed necessary, drainage is established and the artery is dropped back into place. When apicectomy is performed, all the osseous tissue of the apex is removed but is hit with a small curet. The artery is left intact and dropped back into place after the surgery has been completed. No other vessel or nerve comes into the operative field (See Fig. 263.)

This approach is the only route to the posterior or cranial fossa anterior to the internal

In my opinion the procedure of choice in reaching the antecochlear area around the eustachian tube is that devised by Almour. After a radical mastoidectomy has been performed the zygomatic area is eviscerated to its fullest extent, and then a small electrically-driven burr is placed in the tympanic orifice of the musculotubular portion of the anterior external auditory canal wall. The shank of the burr is placed flush with the tegmen tympani, and pressed outward and upward at the upper angle of the wound formed in front by the

remains of the zygomatic root. In this position, the bur enters the apical portion of the pars petrosa anterior to the cochlea and above the

empyema is located within this area, the bur soon meets with pus under pressure, which shoots out through the opening made. The

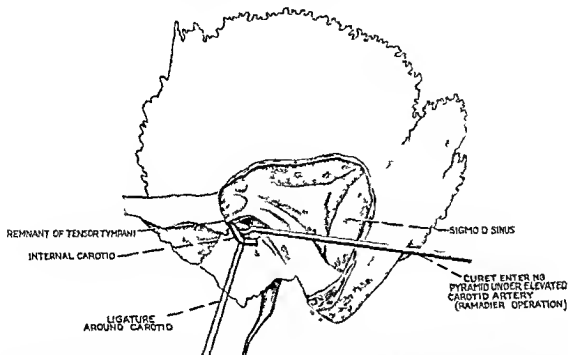


Fig. 263—A loop of gauze or a heavy ligature is carried around the carotid artery after its exposure during the procedure of the Ramadier operation. A curet is carried behind the artery, spicules of bone are removed and entrance into the pyramid is made through the bony bed of the artery. The bony ridge has been left after performance of the radical mastoidectomy as a preliminary step in reaching the operative field. The dura of the middle cranial fossa and the sigmoid sinus are seen.



Fig. 264—Schematic diagram of the Almour interpetrosal approach to the apex of the petrous pyramid. A bur is shown in position above the eustachian orifice and above the carotid canal.

bend of the internal carotid artery and encounters no structures of vital importance in its progress inward (See Fig. 264.) If a closed

opening is then enlarged, a drain inserted, and the wound thereafter handled as that of a radical mastoidectomy.

The Frenckner Operation—There is often a small tract of cellular structures near the tractus subarcuatus which leads under the arch of the superior semicircular canal. Sometimes the tractus subarcuatus is considerably developed with cells and intercellular bone structures. When such a tract is involved in a petrosal suppuration it may be used as a route of evacuation of an empyema which is located in the supracochlear region leading forward from above the cochlea. This method of surgical approach is known as the Frenckner operation. That this approach does not reach the infra-

low and treatment must be directed thereafter to the meninges.

Chronic Petrositis—Chronic petrositis or osseous perilabyrinthitis is a lesion that is often overlooked, and its recognition depends upon making a diagnosis of location in the face of a continuing middle ear suppuration, either before or after a radical mastoidectomy has been performed. Its predominant symptoms are a chronically discharging ear following an acute onset which has abated in intensity, the discharge continuing but not associated with fever or other clinical manifestations.

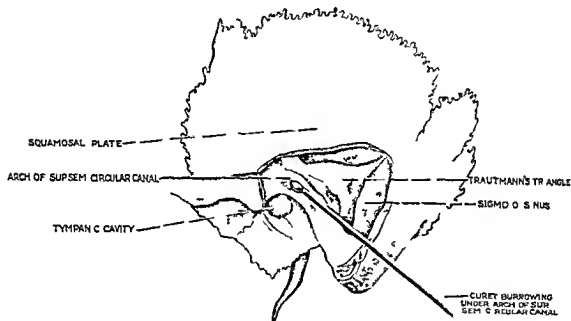


Fig. 265—Frenckner's procedure. After the superior semicircular canal is outlined, a small sized curet is entered below its arch and worked forward and inward making a supracochlear opening posterior to the posterior bony external auditory canal wall.

cochlear regions is the principal objection to its general adoption as a procedure for routine usage (See Fig. 265).

POSTOPERATIVE TREATMENT—The management of the postoperative wound in no way differs from that of any simple or radical mastoidectomy wound. Observation of the patient's temperature, the condition of the sensorium, the local signs and symptoms from the fifth nerve, the relative degree of ocular pain, any change in the hemograms, and the general clinical reaction of the patient give evidence as to whether or not the lesion has been reached and its progress stopped. If its progress has not been stopped, meningeal involvement inevitably fol-

Pathology—In a pneumatized temporal bone an acute otitis media is generally followed by a coalescence of the cells in the mastoid process. The cellular elements in the perilymphatic region also become involved and sometimes a breakdown of the intracellular structures takes place leaving a purulent tract leading into the tip of the petrosal pyramid. Along this tract suppuration continues. Drainage takes place through the middle ear. In the course of the infection in some of these cases an acute mastoiditis develops and presents signs indicating the necessity for surgical intervention. In most cases a simple mastoidectomy is performed. The acute symptoms then abate and usually

the postauricular wound eventually heals, but the middle ear never becomes dry. From the region anterior and superior to, or anterior and inferior to, the cochlea, purulent tracts discharge their contents from the affected regions into the tympanic cavity and from thence, through the perforations in the membrana tympani, into the external auditory canal.

Course—The discharging tracts may continue to present a chronically discharging ear for many years. In many of these cases, generally termed "chronic purulent otitis media," a radical mastoidectomy is eventually performed. In such cases, however, this operation does not result in a dry middle ear because the surgeon, while performing the various procedures embraced in the technic of the radical mastoidectomy, has failed to search for and eradicate the chronically suppurating tracts leading from the petrosal pyramid.

There are no symptoms of intracranial or fifth nerve involvement because there exists, for most of the time, no obstacle to the free flow of the purulent discharge from the petrosal pyramid to the external auditory canal. When interference to this free drainage of the pus from the lesion presents itself, then an acute episode will immediately supervene. Often this will be interpreted as an acute exacerbation of the chronic purulent otitis media, whereas it actually is a blockage of one or more of the afore-mentioned tracts. The acute episode may precede intracranial manifestations. The symptoms from the meninges indicate prompt surgical intervention anent the chronically discharging ear.

Diagnosis—Chronically discharging ears, except those due to an acute necrotic otitis of Witmaack, are not usually found in association with mastoid processes which are pneumatized. In most chronicities of the middle ear, roentgenologic study shows a sclerosed type of bone. Therefore, the history of the onset of chronicity plays an important role in making an estimation of the clinical situation when a case of chronic petrositis comes under scrutiny. The roentgenologic examination will reveal, if the patient has previously undergone a simple mastoidectomy, the remnants of cellular elements still present in some portion of the temporal bone. These may be found in any region of the mastoid process or may actually be demonstrated in the region of the pars petrosa itself with rarefied areas which are actually the seat of a chronic

lesion clearly demonstrable. Such evidences are at times more pronounced when a chronic petrositis presents itself sometime after a radical mastoidectomy has been performed which has not resulted in a dry ear. Here the radical wound cavity very often is found to be entirely epidermatized in the mastoid section of the area operated upon, but, in the perilyabyrinthine region, granulations are present and are usually found around the openings of the suppurating fistulous regions. When these openings are carefully probed they are found to lead anteriorly and inward to the petrosal tip. Thus it is ascertained that the petrosa is the area involved which is causing the persistence of the chronically discharging ear.

Treatment—Treatment again is surgery. All tracts should be searched. Roentgenologic examination may possibly locate the area and when located, direct approach either by the Frenckner, the Ramadier, or the Almour procedure is indicated. My preference is the Almour procedure. If the lesion is left uncorrected, there is usually little danger connected with the chronic lesion, which will continue to suppurate, but life will remain unthreatened.

CHEMOTHERAPY—The sulfonamide compounds have now found their place in the treatment of suppurative lesions. They should not be used, however, as a substitute for essential surgery to eliminate the infective lesion in the bone. Surgical intervention, in acute or chronic mastoiditis or complications thereof, is necessary to remove the herd of purulent matter within the bone before the bacterial static quality of the sulfonamide compounds can exert its greatest benefit.

Therefore, the use of the sulfonamide compounds is strongly recommended following surgical intervention. The dosage should be adequate and the drug should be administered under the control of periodic examination of the blood with estimations of the sulfonamide concentration. The periodic estimations of concentration in the blood should be a guide to the dosage. The withdrawal of the drug should never be sudden. It should be administered in diminishing dosage for at least a week after all symptoms have abated.

When hemoglobin is lost both through the infection and as a result of the chemotherapy, its rapid replacement during the postoperative period is necessary. Repeated small transfu-

sions of from 35 to 50 cc of whole blood should be given every third or fourth day

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OTOGENIC INTRACRANIAL INFECTIONS

The intracranial lesions with which otologists deal, as far as this chapter is concerned, will be restricted to those conditions which develop endocranially as a direct result of prior infections of the middle and the internal ear structures. These prior infections are the lesions which stand in etiologic relationship to those which develop in the adjacent tissues comprising the meninges and the brain itself.

Antecedent Lesions.—The lesions with which we are dealing are of bacterial origin. The character of the invading organism varies with the season and with the type of infection which is prevalent in a particular community at a given time. Local tissue reactions are nearly alike

regardless of the bacterial invader in a given case. Differences due to characteristic specificity of bacterial invaders must be noted, but these do not play the dominant role in determining the course of the pathologic picture. That is dependent upon the bone and its epithelial lining. To this generalization there is one exception. In individuals who suffer from a constitutional or a general debilitating disease, bacterial infections take a more rapidly destructive course. This will happen, for example, in individuals suffering from a tuberculous lesion, or in rachitic children, and also in patients suffering from diabetes.

The tissue reactions are dependent primarily upon the type of tissue involved. Pneumatized bone under bacterial attack produces an osteitis. Diploic bone is apt to produce an osteomyelitis with its accompanying septic type of temperature curve. Sclerosed bone is usually found with chronic lesions. This type of bone resists bacterial attack. These basic bone types should always be noted in the lesions which existed prior to the onset of the intracranial complicating lesion because knowledge of this helps in determining the character of the latter and gives determining data for diagnosis. The complications which ensue after an interval of time in the cases associated with pneumatized bone develop by progression of the inflammatory tissue reaction to infection in the middle ear spaces. Complications from infections lodged in diploic bone usually ensue by invasion through means of the vascular channels. The complicating intracranial lesions which follow infections located in sclerotic bone result from secondary infections and from pressure exerted by accumulated detritus and desquamated epithelium from pseudocystolesteatomas secondarily infected, mostly by saprophytic infections. The intracranial lesion which ensues is produced most often by invasion through perforated anatomical channels that become involved and destroyed as the infection spreads through them. For example, the middle ear infection arrested by the compact bone surrounding it, progresses through the oval or the round window, or it erodes its way into the semicircular canals and spreads from there through the labyrinthine spaces. From the latter it spreads to reach the cranial structures, namely, the meninges and the brain.

Route of Invasion.—Since the detailed history of the antecedent lesion in the middle ear struc-

tures gives a reasonable basis for clinically determining the route which the invasion of the cranial cavity has taken, a careful study of the case history and a review of the roentgenograms of the case from the onset of the illness are of paramount importance. The latter is of considerable pertinence because many of the intracranial lesions have many symptoms in common and it is this study of all the roentgenograms which helps to differentiate one lesion from another, as well as to permit exact diagnosis of the lesion itself and furnish information as to the probable location of the lesion.

Finally, in these major types of intracranial lesions, an involvement of the general blood stream in the infective process must not be overlooked. *Septic thrombi* carried by the blood stream may lodge anywhere within the cranial cavity to involve any of the structures it contains in an infective lesion which eventually terminates in a meningitis.

On its road toward the structures of the brain, larger foci of infections may lodge between the tegmen cellulae, or between the roof of the petrosal pyramid and its covering dura with a collection of pus forming between the dura mater and the bone. This is termed an *extradural abscess*. On rare occasions a collection of pus accumulates between the layers of the dura. This will form an *intradural abscess*. When the pus accumulates anterior to the dura mater, between the dura and the pia, and remains outside of the surface of the brain, it is termed a *subdural abscess*. When the brain substance is involved in a septic focus with purulent accumulations within its boundaries, then there will exist a *brain abscess*, located either supratentorially or subtentorially.

Role of the Cerebrospinal Fluid—The cranial cavity contains the brain and its covering, the meninges, in whose meshes there is a fluid which not only fills the spaces left by the convolutions of brain surface but also fills the space in its ventricles and in the central canal of the spinal column. This fluid is known as the cerebrospinal fluid. Its role is to act as a watery cushion for the brain, and also to carry off from the brain cells the result of brain-cell metabolism. The pressure of the cerebrospinal fluid within the subarachnoid space is kept at a given level, slightly lower than the blood pressure, and this changes only with variations of the heart action and the respiratory rhythm. This fluid filters through the capillaries of the

choroid plexus and the capillaries of the perineural and perivascular spaces of the central nervous system. It eventually reaches the venous blood channels. Chemically, the cerebrospinal fluid in its normal state is in osmotic equilibrium with the blood plasma. Its production is influenced toward an exaggerated production or toward a retardation of production by the relative dilution or concentration of the blood.

When the cranial cavity is invaded by purulent infective material, the biochemical reactions of the cerebrospinal fluid take on changes which vary from its normal reactions. A knowledge of the significance of the normal reactions and the diagnostic significance of abnormal reactions therefore becomes necessary for both the differential and the specific diagnosis of intracranial lesions.

Effects of Increased Intracranial Pressure—The first effect of meningeal infection is a great outpouring of the cerebrospinal fluid from the capillary blood vessels. This results in a demonstrable increase in intracranial cerebrospinal pressure. This increased intracranial pressure is the factor which divides all intracranial infections. One group presents this pressure as the dominant symptom, and the other group does not present a very high spinal fluid pressure, but instead shows toxicity as its outstanding symptom.

The immediate effect of increased intracranial pressure is a compression of the intracranial blood supply, both afferent and efferent vessels being subject to this pressure. As a result there is produced a condition of lessened oxygenation of the parts concerned, namely, the brain tissue. From lessened oxygen tension there results an incomplete oxygenation of carbohydrates with the effect of producing the so called anaerobic type of oxygenation over the aerobic, and large quantities of lactic acid appear in the cerebrospinal fluid. This is a constant finding in meningeal infections. In the normal individual the lactic acid content of the blood plasma and that of the cerebrospinal fluid are about equal. In a person with meningeal infection the amount of lactic acid in the spinal fluid will often be found to be three times as great as that in the blood plasma.

There is a secondary phase of reaction in the development of meningitic lesions. Now the intrinsic cell changes in the brain tissue come into the situation, particularly those of the cells

of the choroid plexus and of the cells of the perineural and perivascular spaces. Cell function becomes hindered. There ensues an effect on cell catabolism which is observable in the spinal fluid content. Choline is present in the fluid in greater quantities than it is normally. At this phase, there is an edema of all the tissues concerned, the lactic acid having changed the iso electric reactions of the fluid and that of the cells bathed by it. Finally, the presence of three times the normal quantity of strong lactic acid in the cerebrospinal fluid results in a decrease of the fluid's alkaline reserve.

In modern nomenclature the actual expression of acidity is termed in common usage the minus logarithm of the actual concentration of the hydrogen ion which is present, namely the pH. In meningeal infection the pH of the spinal fluid is lowered. It must be remembered that a change of 1 in the pH is equivalent to ten times that amount in actual change in the concentration of the hydrogen ion. Thus, any fractional lowering below that of 1 in the cerebrospinal fluid has grave diagnostic significance.

Increased amounts of lactic acid have further consequences. Being a strong acid, lactic acid drives off the carbon dioxide from the carbonates. In meningeal infections therefore, there will be found in the cerebrospinal fluid not only a lowered pH but a decreased bicarbonate content.

There is more chloride in the normal spinal fluid than there is in the normal blood plasma of the same individual. In meningitic patients the excess of chlorides tends to disappear, and blood plasma contents and cerebrospinal fluid contents as regards chlorides are more nearly alike and almost equal in amount. In advanced cases of meningitis the spinal fluid chlorides may drop to as low as 33 per cent of the chlorides found in the blood plasma of the same individual.

Effects of Bacterial Invasion—The presence of bacteria in the cerebrospinal fluid in a meningitic case is of itself of grave significance. The finding of the infecting organism through examination of a smear should always be corroborated by means of culture. The finding of bacteria in the smear gives the clue but the final diagnosis will depend to a great extent upon the result of the culture. Identification of the organism gives a key toward both prognosis and treatment. For example, the finding of a type III pneumococcus in the fluid denotes a

grave prognosis. Until recently the finding of this organism was considered indicative of a fatal outcome. In the last few years, however, cures have been reported. The finding of a tubercle bacillus also indicates a grave prognosis. The presence of influenza bacillus usually indicates a very severe meningeal lesion. Irrespective of the organisms found, however, all bacteria produce similar tissue reactions modified only to a degree by inherent characteristics of their own virulence and toxicity.

The activity of bacteria is generally *fermentive* (acting on carbohydrates) and/or *putrefactive* (acting on nitrogenous compounds). Most bacteria are capable of both kinds of activity. During their fermentative activity, bacteria destroy carbohydrates; putrefactive bacteria destroy protein elements. The terms as here used are limited to the specific action of microorganisms or the enzymes elaborated by them, and represent a fundamental phenomenon of bacterial metabolism. Bacterial growth depends upon the bacteria's own metabolism. During growth, bacteria will destroy carbohydrates, if the latter are in usable form in the media in which the bacteria grow. Usually the effects upon tissues are not harmful just as long as carbohydrates are available in the tissue media. In the normal spinal fluid there is continually present a carbohydrate which is available. When the spinal fluid is invaded by bacteria, the bacteria utilize this carbohydrate and break it down until it gradually disappears. The finding of a gradually disappearing carbohydrate element in the cerebrospinal fluid is of grave significance.

The gradual disappearance of a carbohydrate is noted in successive examinations of the fluid in a patient developing a meningeal infection. Its total absence is found to coincide with the clinical establishment of a meningeal infection. It is a physiological fact that the dextrose in the spinal fluid gives a copper reducing reaction when examined with a Benedict reagent for qualitative analysis. The absence or a reduction in this copper reducing substance must therefore be taken into account with the general findings that have been noted.

Increased Cellular Content—The normal spinal fluid contains but very few cell elements, one to three cells per cubic millimeter. With the advent of a meningitic infection the cerebrospinal fluid cell count rises. With a streptococcal type of meningitis, polymorphonuclear leuko-

cytes predominate. To be noted is the fact that the finding of a moderate increase in the cell count is of graver prognostic importance than the finding of an overwhelming number of cells in the fluid.

Summary—Cerebrospinal fluid examination should include microscopic and chemical studies of samples of the fluid and determination of the pressure under which the samples are obtained. The pressure should be compared with the blood pressure taken at the time the fluid is withdrawn. The physical characteristics of the fluid should be carefully observed. In the chemical determination, a decrease in the carbohydrate content, a lowering of the pH, and a loss of chlorides and carbon dioxide should be noted. The carbonates should be estimated. The amount of lactic acid should be determined and compared with the lactic acid in the patient's blood, drawn at the same time. These studies will furnish the data upon which both a differential and specific diagnosis can be based.

MENINGITIS

Meningitis is one of the many subdivisions of intracranial lesions, among which are included intrameningeal abscess, sinus thrombosis, brain abscess, extradural abscess, suppurative meningitis, and protective meningitis.

Protective Meningitis (Meningitis Sympathetica)—This lesion is most often found following an otitic infection. When pressure necrosis, as a part of the evolution of the pathologic lesion of the acute coalescent type of mastoiditis, destroys the tegmen mastoideum, pus accumulates external to the dura between it and the bone. The abscess actually is in contact with and in continuity with the pus within the mastoid process. Surrounding the abscess formation, the meningeal tissues take on protective reactions to the pus accumulations and limit the spread of the pus.

When the antecedent lesion is recognized as an acute hemorrhagic mastoiditis (influenzal infection), there being present an osteothrombotic phlebitis of the veins of the mastoid process, one expects that an extradural formation of pus is not produced by pus breaking through from within the mastoid process, but rather has resulted from a regression thrombosis to the extradural area, with a breakdown and pus accumulation via the skeletal veins. No break in the bony contours of the mastoid process is generally found. The area surrounding the ex-

tradural abscess presents the evidence of a protective meningitis.

In chronic suppurative mastoiditis of the dangerous type in which bone necrosis extends to the dura and in which purulent fistulous tracts surrounded by areas of hard, ebonized bone lead directly toward the dura, pus sometimes collects extradurally. These pus accumulations in the cerebellar area often take the form of a perisinus abscess when actually they represent a cerebellar extradural abscess surrounded by a protective meningitis. More rarely the abscess forms over the roof of the so called tip of the petrosal pyramid. Its formation in this location is secondary to a suppuration within the petrosal pyramid which has ruptured intracranially.

When a protective meningitis develops around an extradural abscess the removal of the causative factor usually is followed by a subsidence of the signs of the surrounding protective meningitis. Obviously the extradural abscess must also be evacuated.

If dural tissue is destroyed by a purulent lesion which has extended to the intradural spaces the *intrameningeal abscess* which results must be evacuated early lest an ulceration of the brain surface will result. If there is undue delay, suppurative matter may reach the cerebrospinal fluid circulation and a suppurative meningitis will result. Intrameningeal abscesses may be considered as evidences of a localized suppurative meningitis, the localization being induced by the protective meningitis.

Generalized Suppurative Meningitis—This lesion is caused by a diffuse infection of the pia and the arachnoid membrane. The products of the infective process are deposited in the subarachnoidal space. Orogenic suppurative meningitis results from an extension of a suppurative otitic focus. It may also result from the lodgment of metastatic infective material carried by the blood stream.

Diagnosis and Symptoms of Meningitis—As regards to both prognosis and therapy, it is important to determine the type of meningitis present in any given case. In all types of meningitis there are certain systemic manifestations which are the result of the reaction of the body to the invading lesion. The severity of the symptoms varies, depending upon the type of infection, the virulence of the invading organisms, and the resistance of the individual. The toxicity is the result of degeneration of brain tissue.

The evidence of meningeal irritation is practically the same for all types of meningitis. The appearance and severity of intracranial pressure is dependent upon the amount of endocranial tension. High fever, great prostration, anorexia,

signs Vomiting, insomnia, Cheyne-Stokes respiration, cranial nerve palsies, slow pulse rate, increasing blood pressure, and eventually papillary edema and a clouded sensorium are manifestations of the increased intracranial pressure.

DIFFERENTIAL DIAGNOSIS OF MENINGITIS

<i>Protective Meningitis (Meningitis Sympathica)</i>	<i>Purulent Leptomeningitis—Fulminating Stage</i>	<i>Purulent Septicmeningitis—Exudative Type</i>
<ol style="list-style-type: none"> 1 Albumin present 2 Globulin absent 3 Appearance somewhat cloudy 4 Pressure of fluid on first withdrawal markedly increased, but later not so much 5 Copper reduced 	<ol style="list-style-type: none"> 1 Albumin present 2 Globulin present in excess 3 Appearance very cloudy on standing, flocculi may appear 4 Pressure of the fluid on withdrawal markedly increased 5 Copper reduction absent or very faint, as lesion develops, each succeeding test gives less carbohydrates 	<ol style="list-style-type: none"> 1 Albumin present 2 Globulin present in excess 3 Appearance very cloudy, on standing flocculi may appear 4 Pressure of the fluid on withdrawal markedly increased 5 The presence or absence of copper reduction is dependent upon the type of organism present. When the organism is of the type that preferably thrives upon carbohydrates, the copper reduction will be absent early. If the organism is putrefactive, the glucose in the spinal fluid will not always be affected and copper will, therefore, be reduced throughout the disease in some few instances. 6 Cellular elements of the polymorphous type increased, with many disintegrated cells and cells undergoing destruction due to the action of microorganisms. 7 The organism found may be of any group. Most often it is one of the streptococci possessing hemolytic properties. However, staphylococcus, Friedlander's bacillus or B influenzae may be found. 8 Chemistry pH, chlorides, and carbonates are reduced. Lactic acid is markedly increased.
<ol style="list-style-type: none"> 6 Number of cells enormously increased. At first, when the suppurative focus threatens intracranial invasion, the polymorphonuclears predominate. As the body conquers the infection and the danger of meningeal invasion is past, the polymorphonuclear cells disappear and are replaced by mononuclear lymphocytes. 7 Bacteria absent in culture and on smear. 8 Chemistry almost normal. Tendency away from normal (slight rise in lactic acid). 	<ol style="list-style-type: none"> 6 Cellular elements of the polymorphous type increased, with many disintegrated cells and cells undergoing destruction due to the action of microorganisms. 7 Bacteria present in culture and on smear. Usually the organism is found to be pneumococcus of type III, a pure pneumococcus, or Streptococcus pyogenes. 8 Choline present in excess, pH, chlorides, and carbonates reduced. Lactic acid markedly increased over amount found in the blood plasma. 	

and restlessness, with all the signs of severe sepsis, are manifest in all types of meningitis. Meningeal irritation is usually evidenced by rigidity of the neck and spine, ankle clonus, severe headaches, increased deep reflexes, and positive Kernig's, Brudzinski's and Babinski's

There are certain very early signs of meningeal infection which may be present even before rigidity of the neck is noted. A painful sensation upon flexing the head forward upon the chest is one of these. Photophobia, especially when accompanied by generalized headache, is an-

other. Sometimes the very first sign noted is a peculiar abduction and outward rotation of the leg upon the opposite side of the body from the one with the ear involvement. A temporary asynergy in the function of both legs is also a very early sign.

The table of differential diagnostic data (p. 328) is detailed to help in the diagnosis in a given case.

Treatment of Meningitis—In the treatment of meningitis, there are three primary aims: (1) the lowering of the intracranial pressure, (2) the restoration of the alkaline reserve of the cerebrospinal fluid, and (3) the curbing of bacterial invasion.

In an attempt to lower the intracranial pressure and, if possible, keep it within such limits that its action on the vital centers in the brain does not cause death before other remedial therapy has had a chance to beneficially influence the course of the disease, as well as to eradicate possible residual foci of infection within the boundaries of the temporal bone, a complete revision of the surgery on the mastoid process, with or without visual inspection of the middle ear structures, must be undertaken. The structures of the pars petrosa must be carefully studied for evidence of a residual pus foci in them, also the sphenoidal sinus of the same side of the head in which the ear lesion is located needs careful investigation. The possibility of the presence of a purulent labyrinthitis must also be eliminated as a causative factor.

To reduce intracranial pressure which threatens life, a complete decapsulation of the temporal bone, on the side affected, is in order. The tegmen mastoideum, the tegmen tympani, and the roof of the pars petrosa should be removed. Sufficient area of the pars squamosa should also be removed, after retraction of the periosteum and the temporal muscle, so as to permit bulging outward of the dura. Removal of that portion of bony structure which lies backward from the horizontal semicircular canal and extends toward the lateral sinus, comprising the area designated as Trautmann's triangle, should be undertaken, exposing the dura lying beneath it. The exposure should be extended downward and also backward, the entire bony covering of the lateral sinus from the sinodural angle to just near the bony posterior external canal wall being taken away. The bone overlying the sinodural angle should likewise be removed.

Drainage is not the answer to the problem which meningitis presents, and an incision of the dura, under the conditions presented, will not relieve intracranial pressure sufficiently to give practical results. On the contrary, it will only produce a transient drainage, and when intracranial pressure is high, result in a herniation of the brain substance through the incision. Removal of the inner table of the temporal bone, as has been outlined, permits release of pressure from the cerebral blood vessels sufficient to lessen the train of events enumerated at the beginning of this chapter, which were pertinent factors in causing the symptoms of the meningeal infection. As a result of the procedure a better oxygenation of the parts is accomplished, or reestablished balance in brain cell catabolism results, with a better level of pH in the cerebrospinal fluid.

To restore the alkaline reserve of the cerebrospinal fluid, and thus create an environment which tends to hinder the further growth of pathologic micro-organisms and to cause these organisms to restrict their putrefactive action and to revert to their fermentative activity, direct, small, whole blood transfusions are administered. In children 20 to 25 cc. of blood are given every second day, and in adults from 35 to 50 cc. In patients receiving sulfonamide therapy, these transfusions replace the hemoglobin lost through the action of the compound and thus are an added safeguard.

When serial cerebrospinal chemical examinations are made in a patient under treatment, as is my custom, following blood transfusions there is often noted a prompt lessening of the amount of lactic acid present as compared to that in the blood plasma of the same patient, the pH is raised, and there becomes evident also a restoration of the carbonates and chlorides. As a result, the toxic symptoms lessen and resistance is increased. If resistance charts, i.e., charts showing the relationship of the percentage of the polynuclear leukocytes in the blood to the total white cell count, have been kept, a noticeable increase in resistance to the infection becomes evident.

To combat the bacterial invasion specific antibacterial sera or antitoxins and bacteriostatic drugs and chemicals are employed. The sera and antitoxins used will obviously depend upon the micro organism present. Infection due to intracellular meningococcus is best checked with antimenigeal sera. The various types of

streptococci are not materially affected by these sera, nor are generally the pneumococcus of type III and the staphylococcus, but these latter do respond to penicillin therapy. Meningitis due to other organisms can usually be checked through sulfonamide therapy.

Sulfonamide and Penicillin Therapy—In the treatment for meningitis *sulfanilamide* is most effective when the invading organism is a meningococcus or a hemolytic streptococcus. *Sulfapyridine* is of greatest value in combating infection due to Friedländer's bacillus. It is also useful in the influenzal infections, and in pneumococcal meningitis in adults. When a pneumococcal mastoiditis or meningitis occurs in children *sulfathiazole* should be the drug of choice. It may be used in adults as well, and is also effective when staphylococcus is the bacterial invader. The exact value of *sulfadiazine* therapy in relation to given micro organisms has not yet been determined. However, in cases of pneumococcal meningitis in adults, and in meningococcal, streptococcal, and staphylococcal meningitis it has been found effective. My experiences with *sulfadiazine* are too meager to warrant a definite recommendation regarding its use. *Penicillin* has been found effective in combating meningococcal, pneumococcal, staphylococcal, and streptococcal meningitis.

DOSAGE—*Sulfanilamide* dosage varies from 5 to 15 gm. in twenty-four hours. The average optional amount is about 10 gm. For the patient with severe infections who cannot swallow the drug may be given subcutaneously or, if the urgency is great, intravenously, using an 0.8 per cent saline solution. The initial dose should be estimated as approximately 5 to 7 cc. of the solution per pound of body weight in a twenty-four-hour period. As the patient improves and it becomes feasible to give the drug by mouth, the dosage should be estimated at $\frac{1}{2}$ gram (0.04 gm.) per pound of body weight per day. This method of estimating becomes of much importance when prescribing dosage for infants and children.

Sulfapyridine dosage is based upon the concentration of the drug in the blood. When used for intravenous injection 0.01 gm. per kilogram of body weight will produce within an hour a concentration of approximately 1 mg. of *sulfapyridine* per 100 cc. of blood. During the administration of this drug its concentration in the blood should be the guide to the dosage. When the optional concentration is obtained, between 5 and 7 mg. per 100 cc. of blood, then

oral administration is to be continued. The average dosage for an adult weighing 150 pounds is 4 gm.

The following table is helpful in determining dosage.

SULFAPYRIDINE DOSAGE*

Weight of Patient in Pounds	Weight of Patient in Kilograms	Dose in cc of 5 per cent Solution of Sodium Sulfapyridine (1.2 cc per kilogram)	Dose of Sodium Sulfapyridine in Grams (0.06 gm. per kilogram)
22	10	12	0.6
36½	16½	20	1.0
44	20	24	1.2
66	30	36	1.8
73½	33½	40	2.0
88	40	48	2.4
110	50	60	3.0
132	60	72	3.6
146½	66½	80	4.0
154	70	84	4.2
176	80	96	4.8
183½	83½	100	5.0
198	90	108	5.4
220	100	120	6.0

* From 'Ampoules Sodium Sulfapyridine Monohydrate' published by Eli Lilly and Company.

In using the intravenous route, great care should be exercised that none of the drug gets into the tissues about the vein. The injection must be given very slowly. If a vein becomes blocked, another vein at a different site should be employed rather than attempting to force entrance through the blocked vein.

Sulfapyridine produces toxic effects. Drug fever should immediately cause its discontinuance. A rash, due to photosensitivity, is sometimes observed. Exposure to direct sunlight of a patient under *sulfapyridine* therapy must be avoided. Depression, vertigo, dizzy sensations, tinnitus, and episodes of excitement or psychosis are signs of the drug's toxic effects upon the nervous system.

Sulfathiazole is administered to the average adult in an initial dose of 4 gm. Eli Lilly and Company market the drug in $\frac{1}{2}$ -gram tablets. A first dose of $\frac{1}{2}$ grains may be given followed by two tablets of $\frac{1}{2}$ grains every four hours, and continued until there is evident abatement of the patient's symptoms, particularly a reduction in the temperature elevation. Children should be given an initial dose of 0.15 gm. per

kilogram of body weight, up to 25 kilograms, then one fourth of the amount should be administered every six hours. Calculated on the basis of pounds the dosage for children would be 1 gm. for every 15 pounds of body weight up to 60 pounds. This would be followed by one fourth of the initial dose every six hours. The blood concentration of sulfathiazole should be maintained approximately at a level between 3 and 6 per cent. Estimates of the concentration should be made daily. When toxic effects are noted, as evidenced by the appearance of a drug rash, conjunctivitis, drug fever, hematuria, and, occasionally, leukopenia, the drug should be withdrawn.

The initial dose of *sulfadiazine* is about 4 gm., then 1 gm. is administered every four hours until the temperature is normal and the symptoms have been held in check for seventy-two hours.

BRAIN ABSCESS

The occurrence of a brain abscess during the course of or following an aural suppuration is to be regarded as a condition demanding relief.

Aural suppuration, whether acute or chronic, is capable of producing a localized pus collection within the brain substance by direct extension from the ear spaces to the brain or through the medium of the blood stream by metastasis or from emboli from a septic sinus thrombosis. Brain abscess which follows chronic suppuration of the middle ear is produced in most instances by direct extension of the infection of the brain tissue through the destructive action of cholesteatoma.

Brain abscesses, then, fall into the following groups based on the previously existing middle ear lesions:

1. By direct extension
 - (a) From acute coalescent mastoiditis
 - (b) From diffuse suppurative labyrinthitis
 - (c) From chronic mastoiditis of the dangerous type
 - (d) From chronic mastoiditis of the nondangerous type with a superimposed acute infection
2. By metastatic foci
 - (a) From acute hemorrhagic mastoiditis
 - (b) From septic sinus thrombosis

Brain abscesses in general are classified as acute and chronic. This classification is not a clinical one but is based upon pathologic factors and is determined by whether or not the abscess has a capsule limiting its boundaries.

When the abscess forms rapidly and the brain tissue is unable to set up protective barriers against further destruction, no limiting membrane is demonstrable either macroscopically or microscopically, and the abscess is termed acute. Surrounding the abscess is a zone of round cell infiltration associated with numerous hemorrhages and thrombotic vessels. Such an abscess is usually the result of metastatic infection but occasionally occurs through direct extension. The chronic brain abscess usually presents a definite, limiting capsule or limiting membrane. The capsule results from the activity of cerebral protective mechanisms. First there occurs an increase in fibroblastic proliferation which eventuates in the formation of new connective tissue. This tissue limits the progress of the abscess, and accounts for the clinical observation so commonly found in association with this type of abscess, that is, the prolonged period during which symptoms are absent. This period is termed the "latent period" of the abscess. The chronic abscess is usually produced by extension of the infective process from the middle ear spaces. The microorganisms are generally not virulent, and the lesion develops slowly. The initial symptoms are vague, slight, and often not ascribed to the developing lesion in the brain.

The temporosphenoidal lobe of the brain is the most frequent site of otitic brain abscess. The cerebellum is the next most frequent. When brain abscesses are caused by septic metastatic infections they may be located in any portion of the brain.

A brain abscess, if unrelieved through surgical intervention, will eventuate in one of the following ways: (1) It may become absorbed, healing taking place through gliosis. (2) It may empty spontaneously. Its contents may drain off through a fistulous opening through its capsule, or through a pathologic tract opening into the bone and capsule. (3) It may rupture into a neighboring ventricle, or into the subarachnoid space. There then results either an acute encephalitis or a purulent meningitis. (4) The infection, in acute brain abscess, may spread rapidly resulting in an acute purulent meningitis.

Symptoms.—The symptoms of brain abscess divide into those produced by increased intracranial pressure (general symptoms) and those resulting from the destruction of brain substance with consequent loss of function from the injured area (focal symptoms).

There is a group of symptoms attributed to increased intracranial pressure which is in addition due to pus retention. In this group belong headache, chills, vomiting, a subnormal temperature, and the evidence (obtained by examination of the cerebrospinal fluid) of protective meningeal reaction. During the initial stage of abscess formation these symptoms are apt to be obscured by those of disease within the temporal bone. Headache is practically always present in association with brain abscess. Its severity may vary to a great extent. The persistence of headache following surgery in the mastoid process must never be overlooked. In the absence of a classical clinical picture it may be the only symptom of brain abscess. Vomiting, if it occurs early in the course of the development of a brain abscess, is a manifestation of cerebral suppuration. At first it is not projectile, the projectile type occurs mostly when increased intracranial pressure is present. The continuation of subnormal temperature for a period of time, in the presence of the foregoing symptoms, is a definite indication of a brain abscess.

In the type of brain abscess which is the result of an extension of a purulent otitic focus, protective meningitis is present very early in the disease. As soon as the cerebral tissue has reacted to the presence of the purulency within its substance and has to a greater or lesser extent succeeded in localizing the suppuration by confining it within the pyogenic membrane, the meninges outside of the pyogenic membrane form a protective mechanism and the spinal fluid is normal. Later, when the abscess again approaches the meningeal covering, evidence of a protective meningitis reappears in the cerebrospinal fluid. In an abscess which is the result of metastatic infection, evidence of a protective meningitis will depend upon the proximity of the abscess to the meninges. For example, during the very early stage of the invasion it is quite possible to have a normal spinal fluid because the abscess has developed at a point where the meninges are in no danger of becoming infected. As the abscess works its way toward the brain surface seeking egress the meninges immediately react, and a protective meningitis develops, as evidenced on examination of the cerebrospinal fluid.

In the early stages of an abscess extending from a focus within the bone, the spinal fluid will be found to contain a predominance of

polymorphonuclear cells. Later as the abscess becomes localized the mononuclear lymphocytes make their appearance in increasing numbers acting as removers of the detritus of the subarachnoid space. Bacteria are rarely demonstrable in the spinal fluid during the protective stage of a meningitis associated with the formation of a brain abscess. When they are found in the spinal fluid they either are indicative of a suppurative meningitis superimposed upon the already present brain abscess or signify rupture of the abscess into the general cerebrospinal fluid circulation.

The symptoms of increased pressure upon the brain are a slowing of the pulse rate, projectile vomiting, change in the blood pressure, ocular muscle palsies, choked disk, and coma. The appearance of such symptoms always means progression of the disease, in contradistinction to the symptoms previously given which are merely diagnostic of the presence of an intracerebral suppuration. A pulse rate definitely retarded over a prolonged period of time is evidence of the progress of the abscess. Projectile vomiting occurs more commonly in conjunction with meningitis and brain tumors than with brain abscesses. When it is present, however, it is of the usual type and is not as associated with nausea or other gastric disturbances. Abscesses located in the cerebellum are more prone to be associated with projectile vomiting than are abscesses within the cerebrum proper because of pressure exerted on the brain stem with resulting irritation of the vomiting center in the dorsal nuclear of the vagus.

Cerebrospinal pressure is usually just below blood pressure, but with an acute abscess there is generally noted a marked rise in the blood pressure because of the effort of the circulatory mechanism to overcome the obstruction to the free circulation of the blood within the cranium. In the chronic type of abscess in which the increase in intracranial pressure comes on very gradually, no such characteristic rise in blood pressure is noted. There has been time for nature to compensate for the resistance offered by the increased cerebrospinal pressure. When an abscess has reached considerable size and the pressure is exerted against the brain stem, ocular muscle palsies result. The nerves affected are the third and the sixth. When these palsies are not definitely characteristic of brain abscess, they must be interpreted as the result of cerebral compression.

When coma is present, it is well to remember that the prodromal symptoms are produced by the derangement of cerebral function due to the pressure of the abscess. An early manifestation of cerebral compression is a clouding of the patient's intellect. The abnormal deviations in the beginning are scarcely perceptible. Gradually increasing drowsiness appears, alternating with periods of extreme restlessness. The response to stimuli is markedly retarded. This is especially noticeable in the patient's response to questions. There is slow cerebration, and delayed speech in answering questions. Memory also gradually becomes impaired. As the lesion progresses, the patient may be depressed but at times appears irritable, and delirium is sometimes the forerunner of the comatose stage. Occasionally, the patient may become maniacal, necessitating forceful restraint. As the terminal stage approaches the coma develops to a marked degree, with the consequent interference with cerebral function.

Considerable stress has been laid in the literature upon the importance of papilledema in the diagnosis of brain abscess. In my opinion, this is not an important diagnostic symptom. The optic nerve is enclosed in a prolongation of a dural sheath, and this sheath contains fluid in direct communication with the cerebrospinal fluid circulation. The most common causes of papilledema are (1) interference with the return flow of blood from the cranium and (2) an increase in the amount of intracranial pressure due either to an increase in the actual amount of fluid in the subarachnoidal space or to an interference with free circulation of the fluid. In the acute type of abscess where there is apt to be a sudden increase in intracranial tension with obstruction to the general blood supply, the presence of papilledema is rare. In the chronic type of abscess where the signs of increased intracranial pressure are absent, the eyegrounds will be found normal. If the abscess reaches a size large enough to impede the cerebrospinal circulation, or if it is situated in a place where its pressure alone may cause blocking of the cerebrospinal fluid pressure, then changes will be noted in the fundus of the eye. In brain abscess of otitic origin choked disk is rarely found. A degree of papilledema is only occasionally noted. An abscess located within the cerebellum is more apt to be associated with papilledema early in the course of the disease

than are abscesses located in the temporo-sphenoidal lobe.

Local Symptoms of Temporosphenoidal Lobe Abscess.—The temporosphenoidal lobe is one of the areas of the brain which is designated as a "silent" area. Local symptoms from this area generally are absent, and diagnosis must be made from the general symptoms and the findings at operation upon the mastoid. When local symptoms are present, they are of two kinds: those due to the interference with the function of the temporal lobe and those due to pressure upon the adjacent structures. The interference with the function of the lobe is definitely diagnostic in the presence of general signs of brain abscess. The symptoms consist of aphasia, hemianopsia, and partial deafness.

Aphasia is more apt to be present if the left side is involved in a right-handed person. Its presence, however, is difficult to detect when the patient's sensorium is so clouded that the examiner is unable to differentiate between real aphasia and the signs and symptoms of impaired intellect. Aphasia, when present, is generally of the sensory type, that is, word deafness. Inability to name objects is likewise observed as a feature of this sensory aphasia.

In the examination of the eyes a definite search must be made to establish the presence or absence of hemianopsia. If it is present, it is of the greatest diagnostic aid in localizing the lesion in the temporosphenoidal lobe. Hemianopsia is always of the homonymous type.

Since the center of hearing is located within the temporal lobe, one would expect and find a loss of hearing. This loss may not be total because the auditory function is controlled by both cerebral hemispheres, since the ears have a bilateral innervation. It is difficult to determine the amount of loss unless one has on record the hearing acuity before the onset of the primary ear disease.

The symptoms due to pressure of the abscess upon adjacent structures are of two varieties: (1) the motor symptoms which include the palsies, produced by pressure upon the motor areas, or upon the internal capsule; and (2) sensory symptoms which are caused by interference of sensory fibers in the internal capsule. The first sign generally of motor interference is the appearance of a facial palsy on the side opposite to the lesion. The facial palsy is of the supranuclear type which is not

the lower two-thirds of the face, is contradictory to the peripheral type which involves the whole face. As the abscess increases in size and more pressure is exerted on the motor region of the brain, a *weakness of the upper extremities*, on the same side as the facial paralysis, becomes noticeable. This gradually increases until complete paralysis of the spastic type sets in. Gradually involvement of the lower extremities develops and, should the lesion be permitted to develop unchecked, finally *complete hemiplegia* on the side opposite to the lesion develops.

It has been reported that *ptosis* and *dilatation* of the pupil may occur but these conditions are more often associated with brain tumors than with abscess. When the abscess presses upon the retrolenticular division of the internal capsule and involves part of the posterior thalamic peduncle, *contralateral hemianesthesia* develops. Psychological blindness may be present if the abscess involves the posterior third of the first temporal convolution together with the adjacent angular gyrus.

Local Symptoms of Cerebellar Abscess.—It must be noted that a large cerebellar abscess can be present without there being signs of lost cerebellar function. In the early stages of cerebellar abscess, diagnosis depends upon the location of the causal lesion in the temporal lobe and the general signs of brain abscess. The cerebellar abscess hardly ever gives definite diagnostic signs until the terminal stages of its evolution are reached. As a rule definite signs of cerebellar involvement simultaneously appear with the signs of cerebral compression. The signs of cerebellar involvement are *asyn-ergy*, *nystagmus*, and *vertigo*. When present, *asyn-ergy* manifests itself by characteristic ataxic gait. The patient will exhibit a positive Romberg sign, and spontaneous *past pointing* will also be present. *Adiadochokinesis* accompanied by a coarse and irregular tremor and difficulty in mastication is a further sign of *asyn-ergy*. Speech may be slow and interrupted, as such it has been termed "asyn-ergic speech." It is a different type than the aphasia noted in temporoparietal abscess. The *nystagmus* produced by a cerebellar abscess results from interference with the vestibular mechanism of the internal ear. When present it is irregular in character and will be found when the patient looks in any or all directions, very often it is of the vertical type and it may be transient.

Vertigo is always present in association with cerebellar abscess. The sensation of rotation usually seems to be from the affected side toward the sound side. It thus differs from that associated with extracerebral tumors in that the latter produce *vertigo* in which the motion seems to travel from the sound side to the abscess side. *Dysmetria*, due to the disturbance in the patient's judgment of distance and time and also to the force of muscular movement, is noted only in association with fully developed abscesses of the cerebellum. It is demonstrable by the heel to knee test, and by the test in the rapid reversal of the hands. For details regarding these tests, any textbook on neurology may be consulted.

In the search for signs of cerebellar abscesses the functional tests of the vestibular apparatus—embraced by the so-called "Barany test"—have value, but too much reliance must not be placed upon the findings elicited by these tests in one examination only. The tests are of value only if they are repeated as signs of pressure develop. The abnormal *past pointing* following induced labyrinthine stimulation in cases of cerebellar abscesses is always a transient symptom. The character of the reactions on repeated examinations actually constitutes characteristic findings only for a given case of cerebellar abscess. The very bizarre nature of the reactions is due to what Eagleton terms "the establishment of compensation on the part of the cerebellum."

When the abscess causes pressure upon the structures in the posterior cranial fossa and at the base of the brain, the clinical manifestations take the form of *deviation of the tongue* (due to involvement of the hypoglossal nerve in the medulla), *hyperesthesia of the face* on the same side as the lesion (due to irritation of the trigeminal nerve), *facial paralysis*, and *signs of internal hydrocephalus*.

From the foregoing it must be evident that localizing diagnostic data make their appearance late in the course of a cerebellar abscess.

Treatment of Brain Abscess.—*Guides to Surgical Therapy.*—When the findings at the primary operation have shown that the purulent process has reached the tegmen, perhaps to have become localized at this point in the form of an extradural abscess, it is reasonable in the light of a continued syndrome to suspect that the brain abscess is in the temporal lobe. On the other hand, if the lesion in the mastoid and

middle ear leads toward the labyrinth and the spinal fluid shows the presence of a protective meningitis, a persistence in the symptoms indicates first an eradication of the disease in the labyrinth. When the clinical picture shows no improvement after operation on the labyrinth, then the probability of an infection of the posterior cranial fossa is to be considered.

Since the chances for recovery are greatest when the abscess is evacuated early in the course of its development, the diagnosis of the presence of a brain abscess must be made at the earliest possible time. Since, at best, the diagnosis and location of the abscess are more or less problematical, and the operation for the relief of the condition is consequently of an exploratory nature, it is safer to operate and search for an abscess, even though none be present, than to delay operative measures until the disease has passed beyond the stage of surgical relief.

The following principles should be kept in mind in performing an operation for a relief of a brain abscess:

- 1 All operative procedures must be conducted as far as possible in a clean field.
- 2 The barriers erected by nature for the localization of an abscess must not be disturbed.
- 3 Wherever it can be demonstrated that nature has endeavored to evacuate the abscess by forming a tract leading from the abscess cavity toward the outside, the surgeon should employ this tract for drainage.
- 4 The manipulation within the brain substance should be the minimum required to afford surgical relief. The condensation of the brain tissue in the presence of a purulent intracerebral focus is such that any unnecessary trauma may result in spreading the infection, with the production of an acute encephalitis or a severe reaction in the form of a cerebral edema.
- 5 All hemorrhage must be controlled as any extravasation of blood into the subarachnoid space may result in the appearance of compression phenomena due to pressure exerted on the cerebral cortex.
- 6 When the abscess is discovered by a searcher, the latter should be left *in situ* to furnish a guide to further procedure in evacuating the abscess.

Operation for Temporoparietal Lobe Abscess—There are two routes for the approach and the evacuation of the abscess: (1) from the surface of the skull, (2) from the mastoid wound. In otologic surgery the second is the method of choice in all abscesses of the temporal lobe which have resulted from direct extension since it affords drainage through the middle ear and mastoid which, had spontaneous evacuation occurred, would have been nature's method of cure. In addition, it permits drainage by gravity. This is an important fac-

tor, since it is almost impossible to evacuate the abscess cavity completely at the primary operation. The muscular contraction which favors drainage of abscesses located elsewhere in the body is not present in the brain tissue. Consequently, "pump" action is absent and gravity is the only aid. While this operation does not afford a clean field, in the sense that it is performed in an infected area, it is, nevertheless, preferable because the path of extension has been walled off by the protective mechanisms of the meninges, entailing less danger of infecting the subarachnoid space. On the other hand, when the presence of an abscess is unlikely, but when the persistence and severity of the symptoms necessitate an exploratory operation, it is advisable to choose the route first mentioned, as one then operates in a surgically clean area and infection is less likely to be carried into the brain. This is also the method of choice in all abscesses which develop as the result of metastatic infection, whether by an extension of the osteothrombotic phlebitis of the hemorrhagic type of mastoid or subsequent to a sinus thrombosis. These abscesses are usually of the acute type with no limiting membrane and no protective meningeal adhesions. Consequently, operation through the infected area of the mastoid wound is more than likely to cause the entrance of purulent organisms into the subarachnoid space. With a careful observance of asepsis, the operation from the surface of the skull in itself causes less danger to the patient's life and affords less chance of unpleasant sequelae than that through the mastoid process.

No details of the surgery of brain abscess approached through the surface of the skull are here presented. In the most instances these cases are handled nowadays in most of our hospitals by neurosurgeons, not by otologists. Those interested are referred to textbooks on neurosurgery.

OPERATION THROUGH THE MASTOID WOUND—After a mastoidectomy has been performed, it is always advisable to desist from any intracranial surgery unless there is sufficient evidence of cerebral involvement. When the symptoms persist after extirpation of the purulent focus in the middle ear and mastoid process, it is a simple procedure to reopen the mastoid wound and continue the surgery from the point where it had previously been discontinued until the abscess is reached.

In all abscesses which are direct extensions there will be definite evidence of erosion of the tegmen and discoloration and granulations on the dura adjacent to it. When these are found the opening in the tegmen should be enlarged. The granulations on the surface of the dura should be carefully wiped away with a moist gauze sponge and the entire operative field cleansed with peroxide followed by alcohol.

The dura is next incised. The incision is made from before backward, *i. e.*, paralleling the general direction of the temporal lobe. After the dura is opened, all hemorrhage is controlled as far as possible, the edges of the opening in the tegmen are protected by strips of gauze inserted between the bone and the external surface of the dura. This procedure will usually prevent the flow of blood from the mastoid wound into the dural opening.

The pia arachnoid is now in view. When a definite tract is found leading toward the cortex the surgeon should explore along this tract. A large-calibered blunt needle is introduced along this fistulous opening into the temporal lobe, and suction is gently applied by means of a syringe. When pus appears, the needle should be left in situ and a brain knife inserted along the needle until the collection of pus is reached. Drains are then inserted and the knife is withdrawn. The opening in the dura is closed except for the space necessary for the outlet of the drains. The drains should be fixed in position by suturing them to the dural opening.

When no fistulous tract is evident, but when the operative appearance of the meningeal covering suggests the involvement of the cerebral tissue, exploration through the wound should be undertaken. It is accomplished by means of a searcher or by means of a large-calibered needle attached to a syringe. The needle is inserted in the brain substance through the area surrounded by the protective adhesions. It should be inserted directly in the brain and not moved from side to side. If the first puncture fails to reveal the presence of the abscess, the needle is withdrawn and a second puncture is made in a different direction. When the abscess has been located, drainage tubes are inserted in the manner previously described.

Failure to locate the abscess may be due to one of several reasons. Since most of the extension abscesses are of the chronic type wherein a capsule is present, the needle may push the capsule before it and thus fail to enter the cav-

ity. By the use of a needle which, while fairly blunt, is still sufficiently sharp to perforate scar tissue, this error can be avoided. Another common cause of failure in locating the purulent focus is the employment of a needle whose lumen is too small to permit the flow of the thick pus usually found in the chronic type of abscess.

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OSTEOMYELITIS OF THE SKULL

Factors regulating the course and the character of suppurative lesions attacking the marrow-filled cranial bones are

1. The pathway by which the bone is invaded, that is, whether the suppuration results from direct extension of suppuration of tissue within an air-filled space, such as the nasal accessory sinus or the mastoid process, or is brought to the bone by the blood stream, for it may be taken as an axiom that an osteomyelitis which does not start in the neighborhood of an air-filled cavity is of "embolic origin."

Such an osteomyelitis of a bone of the skull is generally a complication of syphilis or diabetes

2 The virulence of the attacking organisms

3 The age of the patient, for the function of the bone marrow depends (in a large measure) on the age of the patient at the time the bone is attacked by infection. For one of the chief functions of the red marrow in the cranial bones is to resist and overcome any infection that reaches it. Furthermore, the morphologic state of the marrow at the time it is attacked dictates the pattern of the osteomyelitic infection. The phosphocalcium basis of the bone acts chiefly as a supporting framework

Osteomyelitis of the Sphenoidal Basis—The spongiosa of the sphenoid occipital cranial basis is filled with red marrow *throughout life*. Thus any pyogenic process of the sphenoidal clavus partakes of the nature of a *sinusoidal osteomyelitis*. Since red bone marrow has great powers of resistance against infection, and when infected, has the power of repair, sinusoidal osteomyelitis of the sphenoidal basis is very rare, and when present, generally subsides spontaneously

In *chronic osteomyelitis* of the sphenoidal basis the temperature of a low grade bone sepsis is associated with pain at the top of the head and chronic congestion of the nasopharynx, which latter is characterized by tenderness upon pressure, and this without pain on swallowing. *Referred segmental suboccipital pain of acute osteomyelitis of the sphenoidal base* occasions pain in the occipital region and the upper part of the neck. This is because the sphenoidal basis develops as a part of the cerebrospinal axis. The location of the pain is due to the distribution of the upper segmental occipital nerves

Osteomyelitis of the Temporomastoid Domain—At birth and during infancy the mastoid temporal domain is filled with primitive lipoidal bone marrow. This is completely replaced by pneumatic cells by the third year. Consequently, an inflammation of the mastoid during infancy is an osteomyelitic process, while after the second year it is an otitis

In the presence of *acute infection of the mastoid in an infant*, the outstanding symptoms, in addition to the otitis, may be severe constitutional disturbances, especially diarrhea, because the gastro intestinal tract, not having acquired its full functional growth, is in an unstable condition

In an infant the usual *subperiosteal mastoid abscess* resulting from a suppurative otitis media is an osteomyelitic process. The abscess is situated above and slightly behind the auricle, because of the position of the tympanic antrum. It is apt to be accompanied by but little pain. When not evacuated the abscess has a tendency to spread forward under the temporal muscle. Simple incision and drainage are usually sufficient treatment because of the reparative properties of primitive lipoidal bone marrow

Acute invasive osteomyelitic phlegmon of otic origin in children is generally of staphylococcal origin. It attacks the mastoid and the bone marrow of the whole auditory domain of the growing temporal bone. The initial otitis media is frequently insidious and attended with little pain but with profound deafness. Embolic infarctions have caused a similar process on the opposite side.

Osteomyelitis of the Forehead from Frontal Sinus Infection—The membrane bones of the cranial vault are filled with red bone marrow throughout life. However, since this red bone marrow is very thinly distributed, an infective osteomyelitic process once well established in the interosseous diploic vessels is not easily eradicated by the action of the red bone marrow

Osteomyelitis of the forehead (metopic osteomyelitis), the result of direct extension from frontal sinus suppuration, is a rarefying destructive thrombotic process. The disease frequently originates in the upper and inner sector of the air sinus, where the bone marrow of the frontal bone may be in direct contact with the mucous membrane of the sinus. The usual type is the *subacute*, which is characterized by a slowly extending suppuration, with a tendency to the formation of diffuse areas of local pus, and of small sequestra of the outer table of the bone. After drainage and partial removal of the infected area the process is apt to remain quiescent for a time, and then, following a cold, slight trauma, or without any known cause, to recur and to advance

The symptoms of subacute metopic osteomyelitis are pain, elevation of temperature, tenderness, and edema, varying in different degrees dependent upon the virulence of the infection and the portion of the bone involved. An important diagnostic sign of an osteomyelitis of the frontal bone is the extension of the

pain which is elicited by pressure beyond the limits of the affected frontal sinus, and its association with headache

In many cases of subacute osteomyelitis the bone marrow of the zygomatic region is primarily attacked and an abscess forms under the temporal muscle above and posterior to the external angular process. The deeply situated subperiosteal abscess is frequently accompanied by but little pain and slight tenderness

Acute invasive phlegmonous osteomyelitis is initiated by a chill or chills, and is accompanied by a high temperature and headache. A complicating leptomeningitis causes early fatality if the infection is not eradicated early by surgery and chemotherapy

The early changes of infective osteomyelitis are microscopic. Superficial (subperiosteal) and deep (vitreous plate) osteomyelitis may be differentiated clinically at an early stage. When the infection is situated in the superficial layer of the bone, the edematous area is moderately painful upon pressure, but when the infection involves the vitreous plate there is exquisite sensitiveness to the slightest touch, while there may be little or no edema. Vitreous plate osteomyelitis is always accompanied by spontaneous pain and severe headache

The chief roentgen ray evidence of acute osteomyelitis of the frontal bone results from absorption of the inorganic salts, which does not occur until between the fifth and the tenth day of the disease. However, there are small vessels situated above the frontal sinus, establishing communication between the bone marrow and the veins external to the skull. These interosseous (diploic) vessels pass through minute apertures in the external lamina of the skull. The thrombotic process may enlarge these apertures, and careful comparison of the size of the apertures on the affected and unaffected side of the frontal bone may enable a diagnosis to be made several days before there are roentgenographic signs of decalcification

When an osteomyelitis of the frontal bone extends intracranially the infection usually causes an extradural abscess, because of the vascular connections that the membrane bones of the cranial vault have with the dura, the bone and the dura developing together in the fetus. Furthermore, since the osteomyelitis is a retrograde phlebotic process, the extradural abscess is apt to be associated with a collection of infected fluid in the subdural space, later becoming

a subdural space abscess. It is apt to be very extensive because there is no mechanism in the subdural space to limit the infection. An intracerebral frontal lobe abscess complicates a considerable percentage of cases of frontal bone osteomyelitis. An osteophlebitis which involves the diploic blood spaces of the vitreous plate may immediately cause a leptomeningitis beginning at some distance from the infecting frontal sinus

Osteomyelitis of the frontal bone requires surgical eradication of the infected area, which usually extends far beyond where it is microscopically discernible, especially in anaerobic or staphylococcal infections. Active chemotherapy should accompany the surgery. In very slowly advancing cases of osteomyelitis of the vault bones the replacement of the collection of infected purulent fluid by a sulfonamide solution has been followed by complete elimination of the disease, even after there is roentgenographic evidence that the bone has been destroyed by the infection

Osteomyelitic Infections of the Petrous Apex from Suppurative Otitis Media—At birth the petrous apex is filled with red bone marrow, which by the end of the second filling out period of growth of the skull's base is replaced by metaplastic lipoidal (yellow) bone marrow. The yellow bone marrow may in turn be invaded by an "overflow" of air filled spaces from the endothelium lined pneumatic cells of the tympanic mastoid domain. However, as long as an apex contains lipoidal bone marrow it has potentialities of metaplasia on the approach of infection, for the lipoidal bone marrow may be transformed into functioning red marrow (metaplasia) and thus again become an active mechanism against infection

Relative frequency of pneumatization of the petrous pyramid as influenced by age is of surgical significance since it dictates the type of the osteomyelitic process and influences the course it will take. Throughout infancy the apex is filled with red bone marrow which actively functions against infection, there is no pneumatization in the apex. However, after the age of six months "tubal-carotid pneumatic cells" (Gordon Wilson) may pass inward from the anterosuperior quadrant of the tympanic cavity towards the carotid canal. After the age of eight years when the spaces of the petrous tip are filled with metaplastic lipoidal bone marrow, air spaces not infrequently reach the semi-

circular canal During the secondary filling out period of growth an overflow pneumatization frequently extends beyond the labyrinth. Pneumatic spaces completely fill the apex in about 35 per cent of the mature apices in adults.

Dangerous cases of aural suppuration may be distinguished clinically from the benign cases (that is those that subside spontaneously)

Later in the disease, when the petrous pyramid is attacked in addition to a continuation of the discharging ear the patient has low grade temperature sepsis and recurrent attacks of pain in the head which occur chiefly at night,² associated with symptoms of blood stream involvement at some period during the illness.

In infants apical infection is an *acute sinusoidal osteomyelitis*. It occasions profound toxic

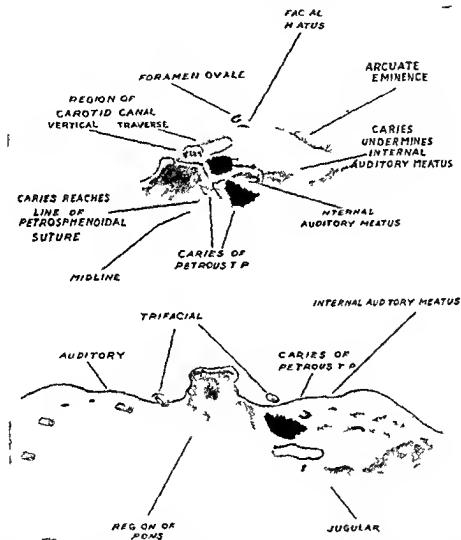


Fig. 266—The extent and limits of the necrosis and cavity formation in a case of suppurative otitis media. The necrosis perforates the cortex in both the middle and the posterior fossa a bridge of compact bone of the superior border being left between them.

Both may present abductor paralysis¹ with retro ocular pain and even hyperesthesia of the homolateral cornea. In the dangerous cases which terminate in apicitis followed by meningitis the otitis is generally initiated by a spontaneous rupture of the drum membrane without severe pain. These symptoms are suggestive of an embolic blood stream origin of the otitis.

symptoms a leptomeningitis rapidly supervening. An unrecognized sinusoidal osteomyelitis is a frequent cause of meningitis in infants. In fact, because of the anatomical position of the apex if suppuration does not subside spontaneously or the apex is not drained intracranial complications most frequently suppurative meningitis will result in all cases. Microscopic

examinations show that a large percentage of all patients with otitic meningitis have apicitis.

The most frequent form of apical infection is a *subacute metaplastic osteomyelitis*. It is most common in childhood, but can occur at any age after the fifth year. It usually originates from slow infective thrombophlebitis of the veins connecting the primary focus of infection in the antromastoid region with the apex. However, the infection may reach the apex by way of any of the tracts of air cells which extend from the antrotympanic domain. In either case there is formed a "granulocarcious tract" situated in the secondary periosteal shell that surrounds the neonatal labyrinth,³ often with areas of pus. The tract is apt to terminate in a localized granulo-purulent abscess, which may fill the apex. The apical abscess, if not evacuated, ultimately breaks through the cortex under the Gasserian ganglion, or into the basal cysterna of the posterior fossa. (See Fig. 266.)

In *insidious (apical) abscess*, caused by a pneumococcal type III otitis media, the infection may have extended by venous pathways into the apex, causing an unsuspected apical destruction, which, if allowed to remain undrained, will result in a fulminating leptomeningitis.

A chronic granulomatous osteomyelitic caries of the petrous pyramid, following an infectious otitis, is apt to be manifested by vague symptoms of "low-grade bone sepsis"—slight irregular temperature, creeping chilly sensation, slight sweating at night, and general malaise. These symptoms may continue for a long time and are frequently disregarded. On the other hand, suppurating foci around the labyrinth may cause signs of labyrinthine irritation, chiefly dizziness. When the infective process is situated near the posterior or superior surface of the pyramid, neighborhood symptoms of meningeal irritation are apt to be outspoken.

Pain in the ophthalmic division of the fifth nerve, limited to the area behind the eye, signifies irritation of the dura over the petrous apex. It is suggestive of caries of the petrous apex.² Venous congestion is apt to cause an associated sixth nerve paralysis, irritation of the dura causes the headache.

Radiographic evidences of disease are discernible only ten days or two weeks after the invasion, when cloudiness of the cells in the angle of the petrosal sinus or destruction of the trabeculae in the petrous tip suggests the pres-

ence of an apical abscess. In the cases of abscess of a fully developed, highly pneumatized apex in an adult, the roentgenogram shows a large cloudy apex and numerous air spaces surrounding the labyrinth.

For details regarding the *operative treatment* of apical infection the section entitled "Suppuration of the Petrosal Pyramid" (p. 318) should be consulted.

In all cases of suspected petro-apicitis the surgeon, before exploring the apex, should carefully search for a fistulous "tract" around the labyrinth. In numerous instances, finding an "extralabyrinthine carious focus" has led to the discovery of the area of apical involvement.

The first stage of "unlocking the petrous pyramid"⁴ allows complete exposure of the external labyrinthine surface. The reflection of the venous lateral sinus backward exposes the posterior surface of the pyramid, including the region of the ductus lymphaticus. The elevation of the dura allows complete exposure of the anterosuperior surface of the pyramid, so that the apex substance can be explored under direct vision. The introduction of a rubber drain, which should be allowed to remain for some time (Ohr's method), will cure the disease.

Lateral pharyngeal abscess may result from petrous osteomyelitic infection. In infants it may be a complication of suppurative otitis from osteomyelitic foci in the temporal bone.⁵ In cases of granulomatous osteomyelitis of the apex it may result from perforation of the abscess into the lateral pharyngeal wall.⁶ All pharyngeal forms of apical infection are controlled by drainage into the throat, simple mastoidectomy, and active chemotherapy.

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PARALYSIS OF THE FACIAL NERVE

Anatomy.—The facial nerve combines motor, sensory, secretory, and vasodilator functions. The motor root arises in the lower portion of the pons, in the region of the nucleus of the abducens nerve. The motor and sensory parts emerge from the lower border of the pons, between the olivary bodies and the inferior peduncles, join the eighth nerve, and enter the internal auditory meatus anterior to the eighth nerve. The facial nerve emerges from the bottom of the meatus, entering the facial canal. This canal proceeds laterally between the cochlea and vestibule, until it reaches the medial wall of the tympanum. The course then turns abruptly backward forming the genu at which point the geniculate ganglion is located. The canal then turns downward between the horizontal canal and the oval window, to emerge from the stylomastoid foramen.

The sensory root has its cell bodies in the geniculate ganglion, situated at the knee of the facial nerve in the facial canal. Central and peripheral branches leave the trunk of the nerve in the internal auditory meatus to form a sensory root. This enters the brain at the lower border of the pons between the motor root and the eighth nerve fibers, then passes into the medulla oblongata, the special visceral sensory fibers ending in the nucleus of the tractus solitarius, and the exteroceptive fibers ending in the descending spinal tract of the fifth nerve.

The motor portion of the facial nerve supplies the stapedius, muscles of the face and scalp, the auricle, buccinator, stylohyoides, platysma, and the posterior belly of the digastric muscles. The chorda tympanica branch carries secretory fibers from the superior salivatory nucleus, special visceral sensory fibers (taste) which end in the tractus solitarius, and

also contains sympathetic fibers to the salivary glands.

The sensory division contains fibers of taste for the anterior two-thirds of the tongue. The nervus intermedius, or nerve of Wrisberg, has as its ganglion the geniculate ganglion. This is connected with the otic ganglion by the small superficial petrosal nerve, with Meckel's ganglion by the greater superficial petrosal nerve which joins the greater deep petrosal from the carotid plexus to make up the vidian, and Arnold's nerve which supplies sensations for the posterior part of the bony wall and tympanic membrane.

Etiology.—Lesions of the facial nerve are central or supranuclear, nuclear, and peripheral. Peripheral lesions are most frequently seen.

Central lesions usually are accompanied by hemiplegia. They may be due to hemorrhage, tumors, softening of the brain, or brain abscess, or they may result from toxic manifestations of such systemic diseases as syphilis, diabetes, diphtheria, or leukemia.

Nuclear lesions are very rare and usually are caused by tumors of the pons. Occasionally they accompany glossolabial paralysis or diphtheritic palsy.

According to Dana, Bell's palsy makes up about 70 per cent of the peripheral lesions. They, too, may result from toxic manifestations accompanying syphilis, diphtheria, diabetes, mumps, influenza, scarlet fever, malaria, tetanus, neuritis, or similar affections. Causes of Bell's palsy are unknown, but exposure to wind and cold, and the so called "rheumatic influence" doubtless are contributing factors. Other causes of peripheral lesions are skull fractures, tumors, and the severance of the facial nerve during operation. In acute otitis media and acute exacerbations of chronic otitis media, the nerve may also be affected by pressure from hemorrhage or edema secondary to infection. Opinions vary as to whether cholesteatoma is in itself a cause of paralysis. Trauma, occurring during mastoid operations and subsequent packing, tumor of the parotid, and other injuries in this region are other causes of peripheral lesions. Ramsey Hunt has described a syndrome in herpes zoster oticus in which there may be an associated facial paralysis. This syndrome also involves herpetic eruptions of the external auditory canal and auricle, pain, tinnitus, loss of hearing, vertigo, nystagmus, and vomiting.

Symptoms—The symptoms of facial paralysis are, of course, dependent upon the cause and site of the lesion. Individuals with lesions above the nucleus are able to wrinkle their foreheads, because the upper branch of the nerve supplying this part of the face has a bilateral cortical innervation, so that lesions of one cerebral hemisphere do not cause paralysis. Peripheral paralysis involving the nucleus or the nerve below causes paralysis of the upper part of the face. A patient thus affected cannot wrinkle his forehead.

to the sound side. Because of this there will be drooling of saliva. The patient is unable to whistle, blow out the cheek, or show the teeth on the affected side. Emotional outbursts of laughter and weeping exaggerate the picture. The nasolabial fold is smoothed out on the diseased side, and, if paralysis persists for long periods of time, there may be some atrophy of the facial muscles.

When the stapedius muscle is involved, the patient becomes unusually sensitive to deep tones, which appear to him to be louder and

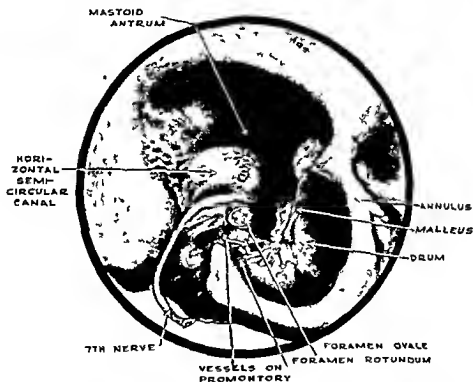


Fig. 267—Dissection showing relations of facial nerve, oval window, and horizontal semicircular canal in an infant.

The onset varies depending upon the cause. Effects may be felt immediately within a few hours or a few days. In Bell's palsy, the attack may be preceded by a tingling sensation or there may be severe pain in the cheek or behind or in front of the ear.

If the paralysis is complete in peripheral lesions, the patient is unable to close his eye, on the side affected. The effort to close the eye may cause the eyeball to roll up, revealing a considerable portion of the white ocular conjunctiva. There may be an associated conjunctivitis with decreased flow of tears. The patient is unable to wrinkle his forehead. The angle of the mouth droops and may be drawn at first

higher pitched than normal. If the lesion lies between the pons and geniculate ganglion there may be a diminution or loss of tears. Loss of taste is noted and salivary secretions diminish when the chorda tympani branch is involved.

Incomplete paralysis is seen in cases in which trauma is a factor. Nuclear and subnuclear lesions are often incomplete. Bilateral lesions sometimes present a 'masked' appearance.

Wilson states that in the chronic stage the flaccid muscles sag; there is a widening of the ocular aperture, until at times an ectropion may result. The corner of the mouth and philtrum are pulled over to the affected side. Contraction of the muscles may evolve with

partial recovery During eating, there may be facial overaction and increased lacrimation Recurrent attacks of Bell's palsy have been reported

Diagnosis—Lesions which are above the nucleus are upper motor neuron type and the upper facial muscles are not affected The tongue may be involved, local reflexes are intact, and there are no reactions of degeneration to the electrical current

Nuclear lesions are rare When present there is usually an associated paralysis of the sixth nerve Hearing and taste are not affected

Lesions between the geniculate ganglion and at the point where the nerve leaves the pons, present lower motor neuron paralysis (peripheral type), disturbance of taste, decreased tears and saliva, and the hearing may be affected (hyperacusis)

Lesions between the geniculate ganglion and the stapedius branch reveal diminished secretion of saliva but lacrimation is not affected There is loss of taste for the anterior two thirds of the tongue

Lesions between the stapedius branch and the chorda tympani nerve cause loss of taste and there is diminished secretion of saliva but hearing is unaffected

In general, peripheral lesions are of the lower motor neuron type When electrical current is used there is evidence of nerve degeneration

Prognosis—The prognosis depends upon the cause and location of the lesion If the paralysis has been caused by operative injury, tumor, pressure, meningitis, or wounds, the prognosis is guarded However, in typical Bell's palsy it usually is good for at least partial recovery, in a period of a week to several months Complete recovery, however, is rare

Treatment—Causative factors determine the treatment of facial paralysis Lesions due to brain abscess or tumors should be treated radically Those resulting from diseases such as syphilis, diabetes, and sepsis should receive vigorous treatment

Patients suffering from Bell's palsy of rheumatic origin receive comfort from heat applied to the area of the ear and parotid region Pain is alleviated with aspirin or anacin Wilson suggests labile galvanism (with anode as the active electrode) over the face for ten or fifteen minutes, once or twice a day He also suggests

weak faradism if the palsy is slight Gentle massage and other methods to prevent the flaccid muscles of the face from sagging also are valuable If contracture develops, electrical treatments should be stopped, but massage may be continued

Paralysis due to acute otitis may respond to myringotomy Following mastoid operation, packs should be removed with the first signs of paralysis Cases caused by infection, or pressure by sequestra or cholesteatoma, may respond to operative procedures

Balance and Duel believe that complete severance of the nerve at the time of operation calls for immediate operative action They describe the following procedures

1 Decompression The facial nerve is exposed from the stylomastoid foramen to the level of the horizontal semicircular canal

2 Direct suture of the nerve ends If the gap is very slight it is possible to suture the nerve ends If the gap is wider the course of the nerve may be shortened by removal from the canal, making suture of the nerve ends possible

3 If the gap is too wide for direct suture of the nerve ends a free nerve transplantation is necessary The anterior femoral cutaneous nerve may be used Return of muscle function may take from six to fourteen months

Anastomosis of the facial nerve with the ninth, eleventh, or twelfth nerve also has been advocated with varying reports of success

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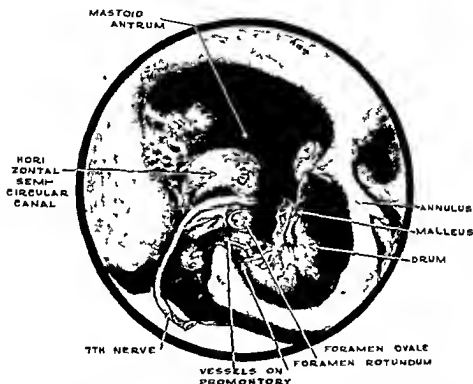


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CONGENITAL AND EARLY DEAFNESS

Terminology.—There exists regrettable confusion regarding the terminology relating to hearing impairment and the classification of hearing deficient persons. To clarify this situation the *Conference of Executives of American Schools for the Deaf*, in 1937, approved and adopted the report of its *Special Committee on Definitions and Standards of Nomenclature*.¹ We quote the following

"1 The Deaf Those in whom the sense of hearing is non functional for the ordinary purposes of life, divided into distinct classes based on the time of the loss of hearing

"a The congenitally deaf

"b The adventitiously deaf—those who were born with normal hearing but in whom the sense of hearing became non functional later through illness or accident

"2 The Hard of Hearing Those in whom the sense of hearing, although deficient, is functional with or without a hearing aid "

These definitions have been approved by the American Otological Society² and other national medical organizations. They emphasize the fact that we should not refer to the hard of hearing as deaf nor indiscriminately use the word deafness to include all degrees of hearing loss. These terms when inaccurately used as sanctioned by long usage imply a certain stigma resented by the hard of hearing and definitely cause confusion.

Etiology.—Goldstein stated "Congenital deafness is that form of deafness resulting from the transmission at birth of a physical imperfection or a pathological process affecting the otic capsule or brain in its embryological development, or some histopathological element of or in the peripheral or central areas associated with the organ of hearing. Congenital deafness, then, may be (1) Biologic, (2) Pathologic."³ In both types heredity⁴ is an outstanding factor. It is noteworthy that more than 30 per cent of congenitally deaf children have some residual hearing⁵⁻⁶ which can be utilized in their rehabilitation. Early congenital syphilis and exceptionally early appearing otosclerosis may be included as forms of congenital deafness.

Acquired deafness is the form of hearing defect caused after birth by local or constitutional disease or traumatism. If the loss is so

profound as to incapacitate the subject for speech contact and occurs before the third year it is as serious and causes equal difficulties for rehabilitation as congenital deafness.

The causes of adventitious deafness include meningitis, influenza, scarlet fever, measles, diphtheria, mumps, and pertussis in the order of the severity with which they affect the hearing. Traumatism, such as basal skull fractures involving the temporal bone or birth injuries causing hemorrhage into the inner ear, produces profound deafness.

In some cases of congenital deafness the function of the static labyrinth is more or less completely retained.⁷

In young children mechanical causes of deafness are hypertrophied adenoids and tonsils, lymphoid hyperplasia in the lateral epipharynx encroaching upon or invading the auditory tubes. Foci of infection in the sinuses, middle ear, teeth, tonsils, and elsewhere in the body are recognized causes of severe hearing loss in children.

Symptoms and Diagnosis.—The outstanding symptom of deafness in the young child is failure to acquire articulate speech at the usual age. This may be noticed as early as the first and second years. Failure to respond to stimulation by loud noises, vocalization, bells, gongs, and other means justifies the belief that the hearing is profoundly affected. The family and the personal history with details as to birth and a thorough otological examination, repeated if necessary, will establish a reasonably accurate diagnosis. Tests with an amplified clinical audiometer yield the most satisfactory results in determining the degree and type of hearing loss. The results of functional hearing tests are not reliable in patients under four or five years unless repeated, and unless cooperation is achieved.

The capacity of the static labyrinth to respond to caloric and rotation tests yields added knowledge concerning the degree of hearing loss and the possible presence of useful residual hearing. Response to loud noises through stimulation of the tactile sense may cause a diagnostic error. Congenital aphasia also may lead to a mistaken diagnosis.

Treatment.—Prophylaxis consists in the avoidance of all known causes of deafness. Individuals known to be capable of transmitting an inherited deafness should be advised to avoid bearing children.

The treatment of deafness in the young is chiefly educational. That the child learn to speak understandably and articulately is the first objective. The child may be taught to speak by modern methods when there is sufficient residual hearing. This is accomplished by early employment of the "Acoustic Method" under especially qualified teachers. The acoustic method is defined by the late Dr. Max A. Goldstein⁵ as "stimulation or education of the hearing mechanism and its associated sense organs by sound vibrations as applied either by voice or any sonorous instrument." This method has superseded the sign, oral and manual methods.

Early enrollment in a residential school or day school for the deaf should be urged by the physician. Time is saved by starting children of preschool age in schools sponsored by the local chapters of the American Society for the Hard of Hearing, established in several cities. Instruction in lip reading and the use of individual and group hearing aids should be prescribed in all suitable cases.

Surgical treatment of the deaf child in selected cases should include the prompt correction of mechanical obstructions which interfere with tubal function, irradiation being employed when indicated. Surgery should be undertaken with the reservation that the procedure will not correct a profound hearing loss, but is executed in the hope of preserving whatever residual hearing is present. The deaf child should be safeguarded by medical supervision against conditions which might lead to greater loss of hearing.

Parents are to be warned against quackery which still thrives in the field of treatment for congenital and early deafness.

Prognosis—The prognosis for any material improvement in the hearing of profoundly deaf children is poor. Rehabilitation, from the educational, economic, and social point of view by the application of modern methods is a brilliant achievement in the care of this group of handicapped children.

HORACE NEWHART

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NEOPLASMS OF THE EAR

The external ear is not an uncommon site of neoplastic disease. As with all skin cancers the rodent ulcers, or basal cell carcinomas, and the epitheliomas, or squamous-cell carcinomas, are the most common types encountered.

From a report by the National Radium Commission, in 2534 cases of skin basal cell carcinomas the pinna was involved in 67, making an incidence of 2.7 per cent. From the same report, in 952 cases of epithelioma of the skin the ear was most commonly affected, the incidence being 20.7 per cent, or 197 cases.

Although any part of the external ear may be involved, in men most of the lesions occur on the upper border or the posterior surface of the pinna while in women the concha and external auditory canal are the frequent sites of involvement.

The temporal bone may be the site of primary neoplastic disease or it may be involved by secondary extensions from contiguous structures. When it is involved by a primary lesion the site of origin is the middle ear, the petrous pyramid, or the mastoid process. New growths involving the skin of the mastoid process may invade the underlying mastoid cortex and extend into the cells of the mastoid process. Likewise, lesions of the concha and external canal can by direct extension reach the middle ear, either through a previous perforation of the tympanic membrane or by direct involvement of the drum. When a neoplastic lesion is found medial to the isthmus of the external canal, its site of origin is extremely difficult to determine. At times a growth whose primary site of origin is within the middle ear cavity will, by direct

extension, perforate the tympanic membrane if no previous perforation existed, or grow through the perforation, if one did exist, and present itself in the external auditory canal. A new growth of the external auditory canal medial to the isthmus of the canal is a potential middle ear lesion and should be treated as such. A primary new growth of the middle ear cavity may likewise spread by direct extension to the mastoid process and the petrous pyramid.

Secondary involvement of the temporal bone may also occur by direct extension from primary lesions of the nasopharynx, the dura, the acoustic nerve, and, not infrequently, from primary lesions of the parotid gland.



Fig. 268 —In the male the pinna is a frequent site of cancer

Symptoms—It is quite evident that in a region containing as many anatomic structures as the temporal bone the symptoms of neoplastic disease will depend upon the location of the lesion and the extent of its growth. It is unfortunate, as with cancer elsewhere, that the early lesion will in many cases present so few symptoms that the tumor is oftentimes overlooked. On the other hand, an early growth may by its location involve a vital structure and, although the new growth is small, produce marked symptoms.

The symptoms suggesting tumors of the middle ear or metastasis extending to the middle ear are (1) pain, (2) discharge, (3) hemorrhage, (4) facial nerve paralysis, (5) deafness, and (6)

vertigo and nausea when the labyrinth is involved.

In cancer of the middle ear and mastoid the pain may be referred not only to the ear but to the mastoid, the temporo occipital, or frontal areas. It is usually of a dull, steady aching character, occasionally lancinating, although it may be only a sense of pressure. At times the pain is worse with cessation of aural discharge and occasionally it appears worse at night.

Although it may have but a casual relationship, chronic discharge is a frequent symptom. The otorrhea may be of a watery consistency, scanty or profuse, at times very foul. Whether the irritation of a chronic suppurative process is an activating factor in aural new growth, the association is of sufficient frequency as to suggest such a possibility.

Hemorrhage from the external auditory canal is suggestive of aural malignant disease. The bleeding may range from a blood-tinged discharge to an excessive hemorrhage following the removal of a specimen or a growth mistaken for an aural polyp.

Deafness usually occurs early and in the early lesion may be the only symptom. It is of the conduction type and can be easily mistaken for tubal obstruction. With the increase in size of the growth and with extension through the tympanic membrane, extension into the external auditory canal takes place and the diagnosis can be established by biopsy.

Involvement of the facial nerve, manifest by *facial paralysis*, is at times an early symptom. However, in many cases, depending upon the location of the lesion, the facial nerve either in the middle ear or in the fallopian canal may not be involved, or it may become involved only late in the disease.

Vertigo and nausea signify involvement of the semicircular canals through erosion by direct extension of the new growth, with consequent labyrinthine disturbance. Fortunately, the canals are involved infrequently and when they are it is usually late in the disease.

Diagnosis—Tumors of the middle ear and mastoid process are no longer considered medical curiosities. A cancer-conscious otologist is the prime requisite for an early diagnosis. When the growth extends into the external auditory canal biopsy is easy and confirms the diagnosis. All aural polyps when removed should be examined microscopically. Any aural polyp that bleeds excessively upon removal

should immediately raise the suspicion of cancer and failure to section the specimen is inexcusable. Incision of the tympanic membrane to obtain a biopsy specimen is justifiable.

There are no characteristic findings by *roentgen ray examination* in early cancer of the mastoid and middle ear. When the growth originates in the middle ear roentgenograms show no abnormality until bone is destroyed. From the middle ear the growth may extend into groups of mastoid cells and yet fail to produce sufficient destruction to be detected by the roentgenogram. In angiomas of the middle ear and mastoid, however, Hampton and Sampson describe rather characteristic findings: (1) an increase in



Fig 269—Angioma of the middle ear and mastoid showing (1) enlargement of the emissary vein (2) enlargement of the external auditory meatus, (3) destruction of the mastoid cells

the size of the vascular channels of the skull on the affected side, (2) enlargement of the groove and foramen of the mastoid emissary vein, (3) erosion of the petrous pyramid, and enlargement of the internal and external auditory canals and (4) destruction of the mastoid cells.

Treatment—In the treatment of cancer of the ear, radiation, electrodesiccation and surgery each have a place. When the growth involves the pinna and is well localized so that removal can be accomplished through a wide margin of normal tissue, surgical removal followed by skin grafting and reconstruction perhaps is the best therapy.

New growths of the petrous pyramid with cranial nerve involvement are beyond the realm of present day surgery. Their treatment is en-



Fig 270—New growth involving the middle ear with bone destruction

tirely by irradiation and, since many of these growths are of the anaplastic type, improvement under irradiation may be striking and complete cure not impossible. As with many of



Fig 271—Destruction of the middle ear by cancer

the radiosensitive tumors, after primary improvement and regression of the growth, recurrences are frequent and fatal termination ensues.

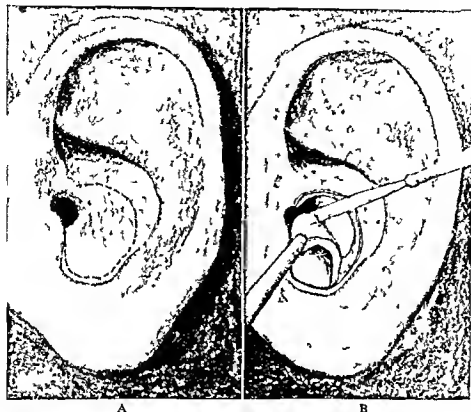


Fig 272—A In cancer of the external auditory canal the entire canal and concha are included in the surgical removal B The incision is carried to the bone by the diathermic knife and the perosteum elevated



Fig 273

Fig 273—Hemangioma of middle ear and mastoid thirteen years after operation



Fig 274

Fig 274—Hemangioma of the middle ear and mastoid two years after operation

A growth of the skin over the mastoid should be removed by electrodesiccation through normal tissue. Any suspicion of extension to the underlying cortex calls for a radical mastoidectomy

to be followed by local application of radium or radon seeds and external irradiation.

When the growth involves the concha and external canal, the entire concha and canal

should be removed and a radical mastoidectomy performed to insure that the mastoid cells are not involved

Involvement of the middle ear presupposes involvement of the mastoid cells, and a radical mastoidectomy is indicated. Only by a radical mastoidectomy can the exact extent of the new growth be determined. The growth is removed surgically as thoroughly as possible. Electrodesiccation is of great value in destroying the growth and facilitating its surgical removal. Surgery also provides adequate drainage of the ear. The radical cavity is packed lightly with gauze and in the center of the gauze a nest of radium needles or radon seeds is placed.

Depending upon the type of growth and its extent, radiation of 600 to 4000 mg. hours is delivered to the area. The postaural incision is left open. Additional external irradiation to the limit of skin tolerance should supplement the local radiation.

LEROY ALLEN SCHALL

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INJURIES OF THE EAR IN WAR

Wounds of the External Ear.—Tears, cuts, and lacerations of the pinna require repair similar to that of other parts of the head and face, survival of tissue, even if almost detached, is probable owing to the good blood supply. Perichondritis is liable to complicate infected wounds and burns and is difficult to treat. Sulfanilamide powder, applied early, is of value, but if chondritis threatens to spread, our advice is to excise the affected area of cartilage, without sacrificing skin.

Injury to the External Auditory Meatus.—Foreign bodies, such as pieces of metal, fragments of brick or pebbles, are removed with a small hook such as that used for clearing cerumen. Syringing must be forbidden, for fear of traumatic communications with the tympanic cavity or interior of the cranium, the driving in of lotion would have the danger of introducing infected material.

Lacerated skin edges are trimmed and purified, but are not widely excised, obstruction of the meatus may require restorative operations to prevent or remedy stenosis, probably with the introduction of a skin graft, held in place by a hollow obturator.

Rupture of the Tympanic Membrane.—Blast, insufficient to rupture the membrane, may cause dilatation of vessels or effusion into the tympanic cavity, with corresponding signs and symptoms. A more severe injury, common in both field warfare and in air raids, is rupture of the tympanic membrane as the result of blast. The individual may be unaware of the aural injury, as the bursting of a bomb nearby not only distracts the attention but is also liable to inflict other injuries of greater moment. The wearing of ear defenders is effective as a means of prevention, but is objectionable for other reasons. Sudden pain may be experienced when the membrane bursts, but often the first intima-

tion of the accident is the discovery of blood in the meatus

The perforation involves the inferior segment, more often in its anterior region, it is unlikely to be marginal and has clean cut edges, with at first but little reactionary change. To avoid missing such conditions, it is necessary to examine the ears in all cases of injury caused by blast, the agent may be an aerial bomb, the muzzle blast of a gun of relatively small caliber—particularly 3.5 cm—and sometimes an explosion in a gun turret. Symptoms and signs, other than those of pain and hemorrhage, are deafness, tinnitus, and occasionally otorrhea, vertigo and nausea may be noticed, but in a minority of cases.

The deep meatus is normally sterile, and if infection is not introduced from without the blood will absorb and the perforation heal, if it is not very large. With this in mind, treatment is first directed to the removal of cerumen and debris by means of a hook or scoop under direct vision. The pinna and entrance of the meatus are then mopped with biniodide of mercury—1 part in 500 parts of alcohol. No antiseptic is introduced into the deep meatus unless debris has penetrated this far, in which case, mopping with a wool-tipped probe, moistened with spirit, is all that is desirable. Syringing is forbidden. A sterile plug of wool is kept in the purified meatus for several days. Any nasal or postnasal infection is treated and the patient is warned against blowing the nose vigorously. The majority of patients thus treated will recover without infection and without marked loss of hearing, rarely a cortical mastoid operation is required for subsequent infection with continuation of suppuration.

Penetrating Wounds of the Tympanic Membrane.—In uncomplicated penetrating wounds of the tympanic membrane, treatment is similar to that described for rupture of the membrane. However, if there is an associated laceration of the external meatus, or penetration or fracture of the surrounding bone has occurred, an operation is necessary for removal of foreign

bodies and bony fragments and for cleansing of the wound, which is best accomplished with alcohol followed by the insufflation of sulfanilamide powder.

Copious discharge, if it occurs, is treated by the instillation of drops of perchloride of mercury (1:4000 in glycerin) after thorough cleansing, as discharge lessens, alcohol is used and, later, sulfanilamide powder.

Wounds of the Mastoid Process.—Penetrating wounds in this region may be associated with injuries of the external ear or meatus, tympanic membrane, or internal ear, each of which requires appropriate treatment. Shock and unconsciousness may be present, followed by vertigo and vomiting. As regards the mastoid itself, early operation is indicated to remove loose bone, foreign bodies, and debris and to give free drainage in obviously infected cases. Sulfonamide powder is dusted onto all parts of the wound, which is closed only if no suspicion of infection is present. No operation for exenteration of the air cells should be performed at this early stage, this should only be carried out if infection continues overlong and then not before the lapse of fourteen days.

Owing to the serious possibilities in injuries of this type, not only as regards local infection but also from the danger of intracranial complication it is wise, in a recent case, to give a prophylactic course of penicillin, or sulfathiazole—1 gm. every four hours for three days.

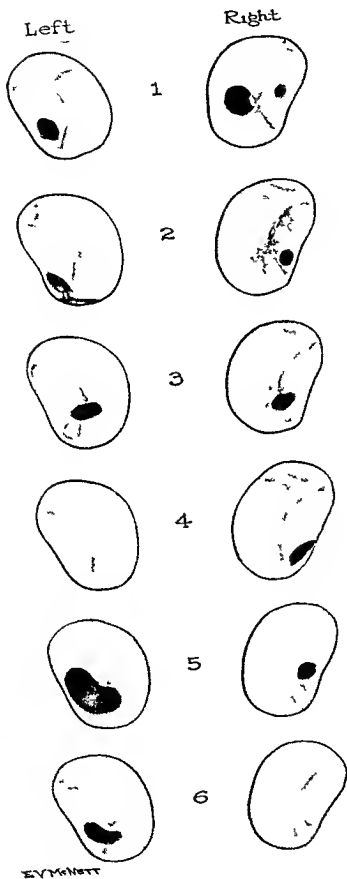
Injury to the Facial Nerve.—Injury to the facial nerve is a complication which does not differ materially as to treatment from that of peacetime cases. In extensive injuries the first aim is to secure the safety of the patient, when all chance of infection has disappeared, decompression of the nerve or insertion of a graft may be required.

Fracture of the Base of the Skull.—This type of injury may involve the ear, the fracture lines having a liability to pass through the roof of the bony meatus, the tegmen tympani or antrum, or through the internal ear. The greatest danger is infection of the meninges, since the fracture

Fig. 275—Traumatic rupture of tympanic membrane. The illustrations have been made from patients seen a few hours or a day or two after injury by blast. The perforation is usually nonmarginal with clear-cut margins and little reaction in the surrounding area. Some of the drawings suggest a rolled-over edge of the torn membrane as the result of the phase of rarefaction.

In 2 (right) there are deposits of dark blood on the membrane, sometimes these hemorrhages obscure the site of perforation.

In 2 (left) and 4 the tear has reached the tympanic ring, the region being situated over the eustachian orifice and therefore unsupported.



E. V. McNett

Fig. 275

may be associated with tearing of the dura mater and the provision thereby of a route of communication with the exterior. A fracture of the base, often of no great intrinsic consequence, thus becomes a potentially dangerous injury. Blood appears in the external meatus and its origin will be in doubt, unless undue curiosity is shown. To be on the safe side, it is wise to carry out only those methods of purification already detailed, covering the ear subsequently with a sterile dressing and bandage. Any interfering attempts to remove blood by syringing, or to attain asepsis by the instillation of drops, are to be deprecated. A prophylactic course of penicillin or sulfathiazole is given for at least three days and sometimes for one week.

If cerebrospinal fluid escapes alone or mixed with blood, the diagnosis is of course beyond doubt, but the treatment is similar to that described. Most clefts close spontaneously but if spinal fluid continues to appear after one or two weeks, an operation may be required to find the cleft and to close it with a muscle or fascial graft, especially if infection is present in the middle ear. An operation can be performed either by the mastoid route or, if preferred, in uninfected cases above the level of the middle ear.

Fracture through the internal ear produces shock, followed by severe vertigo and complete deafness. Treatment is mainly directed to the prevention of septic invasion.

Paralysis of the facial nerve is a possible result of simple fracture.

Injury to the Internal Ear—The internal ear is seated so deeply that cases of direct penetrating injury are seldom seen in the hospital. The dangers of death from penetration of the cranium or from meningitis are considerable.

Occasionally cases of blast injury are found to show cochlear changes of varying degree and duration. Usually, however, the brunt of the injury is borne by the tympanic membrane. Single explosions are unlikely to disturb the labyrinth and it is to repeated high pitched noises that most cases of cochlear disturbance are attributable. The prognosis depends on the degree of hearing loss. The treatment is inactive and expectant, unless tinnitus is severe, when luminal may be useful.

Intracranial Complications—There is nothing peculiar about the complications caused by spread of infection in traumatic conditions of the ear: the possibilities are extradural abscess

localized or diffuse meningitis, encephalitis, and brain abscess. In the first case, thorough exposure of the dura is required with local and oral administration of a sulfonamide or injection of penicillin to prevent extension. In meningitis and encephalitis, the concentration of the sulfonamide in the blood and cerebrospinal fluid must be maintained at a high level, usually by intravenous administration. In these conditions as well as in brain abscess, any operation must be circumspect and so designed as to avoid spread of infection.

V E NEGUS

FUNCTIONAL EXAMINATION OF THE LABYRINTH

The authors describe in this article certain simple tests which if properly applied will give some information about the labyrinth. This end organ is extensively connected throughout the nervous system and a great deal about it is unknown. An attempt has been made to present this subject in such a way as to stimulate the students' interest. In order not to discourage the novice, the more complicated tests and detailed information have been avoided. Listed at the end of this chapter are a number of research reports and reviews to which the reader may refer.

Description of the Labyrinth and of Labyrinthine Function—The human labyrinth contains the three semicircular canals (Fig. 276) and two otolithic organs, the utricle and the saccule. The lagena and other otoliths which are described in lower animals are not present in man.

The Semicircular Canals—Meniere in 1861, suggested that a lesion of the semicircular canals might be the cause of dizziness. Goltz (1870) was the first to appreciate that the function of the semicircular canals is to maintain equilibrium. About 1875, Mach, Breuer, and Crum Brown, quite independently of one another, advanced a hypothesis to explain the mechanism of semicircular canal stimulation. They suggested that it is the flow of endolymph through the ampulla which stimulates the crista and its nerves. Fifty years later Magnus and de Kleyn described postural and righting reflexes and showed their relation to labyrinthine

reflexes Tait and McNally, in 1925, showed by uncomplicated ampullary nerve section in the frog that a semicircular canal elicits reactions of the head and body musculature necessary to compensate for any head movement in the plane of that canal. They also showed that an unopposed semicircular canal in the frog elicits an excessive compensatory reaction. They classified the semicircular canals into two groups: (1) a nongravity set—the horizontal canals—which are concerned with turning movements

with cupular movement or deflection. Ross recorded action currents from the individual nerves of the semicircular canals in the frog and confirmed the theory that the semicircular canals are stimulated by angular acceleration. He showed that it is deflections of the cupula that are signaled to the brain and that are responsible for the nystagmus resulting from stimulation of the semicircular canals.

The Utricle—The utricle is the most important gravity organ in the labyrinth. It is stimu-

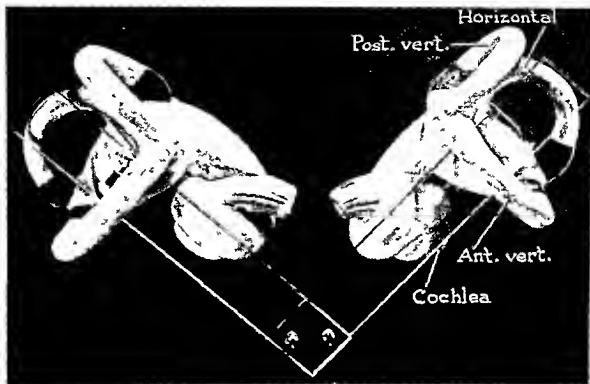


Fig. 276—The model shown was devised and constructed by E. R. Arellano. It represents both internal ears. The glass slide, passing through each internal ear, represents the petrous pyramid. Note that the posterior vertical canal is in the line of the petrous pyramid, whereas the anterior vertical canal is at right angles to it. The picture is taken looking from above. Where the two glass slides join is the front of the head. The ampulla at the end of each semicircular canal is painted black. Passing from the base of each cochlea toward the mid line is the stump of the eighth nerve. Note that the ampullae of the horizontal canal and the anterior vertical canal are together and forward, whereas the ampulla of the posterior canal is behind and below and is not near the other two. The otoliths are not shown in this model.

about a vertical axis, e.g., waltzing. (2) a gravity set—the vertical canals—which are concerned in leaping and landing again, in protecting against falling, and in any movement about a horizontal axis in which the limb muscles are exerted in opposition to gravity. Steinhausen, in 1927, demonstrated cupular movements in the live pike and thus proved conclusively Mach, Breuer, and Crum Brown's theory of ampullary function. His experiments have associated nystagmus during and after turning

lated by slow tilting about a horizontal axis, by centrifugal force, and by linear acceleration. It elicits all the positional reflexes of the body and eyes, previously allocated to both the utricle and the saccule.

There is some experimental evidence to indicate that the utricle responds to sudden movement about a horizontal axis, that it elicits an anticompany reaction which opposes and serves to control the compensatory reactions from the semicircular canals.

Tests for utricular function in man have not sufficient experimental support to be considered in this brief survey

Muscle Tone and the Labyrinth—Ewald observed that a pigeon after a bilateral labyrinthectomy showed two types of tonic disability (1) It lacked capacity for acting quickly and this has been shown by Tait and McNally to be due in large part to absence of the semicircular canal reflexes (2) Its head tended to be displaced when subjected to steady pull or pressure. If the eyes were covered the head assumed an unnatural position which action was followed by changes in limb posture. Tait explained that this second disability occurred because the neck reflexes described by Magnus and de Kleyn were affected by the abnormal head position and they in turn initiated changes in limb posture.

The stretch reflexes of muscle and the tendon reflexes described by Liddell and Sherrington are in themselves unable to protect an animal without labyrinths in a sudden emergency such as landing after a jump. They are only local reflexes. It requires the rapid message from the semicircular canals to the whole body musculature to prepare the animal for a sudden muscular emergency.

Tait and McNally have analyzed the postural changes which follow unilateral labyrinthectomy. There is an asymmetric forced lean to the side which has been operated on for which the removal of the utricle is in large part responsible. It has been conclusively demonstrated that removal of any individual semicircular canal imposes a characteristic residual pose on the animal. It is not a forced posture in that the animal is free to assume any other pose whatsoever but the characteristic residual pose attributed to the absence of that particular canal is the more frequent.

The semicircular canals and the utricles are both concerned in the maintenance of postural tone.

The Sacculle—Most experiments indicate that the saccular otolith has no vestibular function. Evidence is accumulating to show that it may be a vibration receptor possibly connected with the function of hearing.

Nystagmus—It has been shown by Magnus and de Kleyn and others that the only part of the central nervous system essential for the production of the slow and quick phases of nystagmus is that part of the brain stem situated

between the oculomotor nuclei above and the vestibular nuclei below. Other parts of the brain may exert a profound influence upon the nystagmus but they are not essential for its production. Lorente de No and Spiegel have shown that the posterior longitudinal bundle and the substantia reticularis both carry fibers from the vestibular to the ocular nuclei and that either may be severed without abolishing the reflex. Lorente de No believes that there is a rhythmic center situated within this area the intactness of which is essential to the existence of the quick phase of nystagmus. Spiegel on the other hand does not consider such a center necessary and suggests that both slow and quick phases have their origin in the vestibular nuclei and that the rhythm is dependent upon inhibition and refractory phase of the nerves concerned.

Nystagmus and the Utricle—References have been made in the literature to nystagmus resulting from utricular stimulation or disease but there is no experimental proof that the utricle is concerned in the production of nystagmus. On the contrary the centrifuge experiments of Magnus and de Kleyn in which the otoliths were removed from the utricle and the sacculle of the guinea pig the ablation experiments of Versteegh on the utricular macula of the rabbit and the direct stimulation of the utricular macula in the pike by Ulrich did not cause nystagmus. These different experimenters noted that if any injury to the semicircular canals occurred during their experiments nystagmus immediately supervened. For the above reasons we will not devote further attention to the possibility of nystagmus arising from utricular stimulation.

Types of Spontaneous Nystagmus—Spontaneous nystagmus may be caused by disease or irritation of any of the structures concerned in the normal reaction. Three types are recognized clinically: central nystagmus, ocular nystagmus and vestibular nystagmus.

In distinguishing between these three types of nystagmus it is important to note that the first two are usually of long standing. Central nystagmus is usually associated with other signs of intracranial disease. Ocular nystagmus is frequently a to and fro movement without a definite slow and quick component and is usually associated with other signs of disease. Vestibular nystagmus is relatively of short duration—a matter of weeks. At first it is directed

towards the side of the lesion and finally towards the opposite side. It is usually associated with dizziness and some loss of hearing.

Features of Nystagmus—Fischer and Wolfson suggest that in studying or recording nystagmus, the following seven features should be noted (1) whether or not movements of both eyeballs are identical, i.e., associated or dissociated, (2) the form of the nystagmus, e.g., horizontal, rotatory, etc., (3) the direction of the nystagmus, (4) the degree of nystagmus (he describes a first, second, and third depending on how strong the nystagmus is), (5) the excursion of the movement, i.e., the amplitude, (6) the number of movements per second or per minute, i.e., the frequency, (7) the duration of the nystagmus.

Direction of Gaze during Labyrinthine Tests

—When spontaneous nystagmus is present a careful search should be made for some point at which the eyes are steady, before any labyrinthine tests are done. The patient should then be directed to gaze in that direction during the test. The nystagmus resulting from the test stimulus can then be more accurately assessed.

Types of Ocular Nystagmus—Two types of ocular nystagmus likely to be confused with labyrinthine nystagmus are (1) fatigue nystagmus which occurs in a debilitated patient who is forced to keep his gaze fixed to either side for too long a time, (2) fixation nystagmus which occurs when a patient, who has slight difficulty in fixing his gaze, first looks to either side. Other types of ocular nystagmus, including optokinetic nystagmus (train nystagmus, etc.) will not be discussed.

Observation of Nystagmus—Observation of nystagmus is greatly facilitated by using high-powered convex lenses as suggested by Bartels. Several modifications of these glasses are available, including one with lights attached to the frame between the glasses and the eyes (Frenzel). This lighting improves the observation of the nystagmus and with the observer in relative darkness, the patient's fixation is almost completely eliminated and finer degrees of eye movements can be detected. These glasses however, are not essential for an ordinary clinical test.

Clinical Investigation—An examination of the labyrinth is only one essential part of an ear, nose, and throat examination. The history of the case should contain a complete description of the onset and character of the symp-

oms. It should contain an accurate description of the patient's dizziness. Examination of the ears, nose, and throat should include hearing tests and catheterization of the eustachian tubes to establish their patency. The final diagnosis of a labyrinthine abnormality should be made only after a complete physical examination.

The object of the labyrinthine tests is to determine the responses of a certain labyrinth to stimulation. Stimulation causes nystagmus of the eyes, past pointing of the arms, and subjective sensations of vertigo. These will be used as an index of labyrinthine response to stimulation.

Spontaneous Phenomena—The eyes should be examined for the presence of spontaneous nystagmus before any labyrinthine tests are carried out. The head should be placed in different positions, first, to see if there is nystagmus in one position and not in another, and secondly to see if any spontaneous nystagmus is affected by a change in the head position.

The head should be moved suddenly in the plane of each semicircular canal to determine whether nystagmus or subjective sensations result. At this time the presence or absence of Romberg's sign, the ability to stand on one foot, and the ability to walk a straight line should be investigated. The routine cerebellar function tests, such as those for incoordination, sense of position, and adiadokocinesis, should be given.

Induced Phenomena—**THE ROTATION TEST**—The earliest clinical test of labyrinthine function was the rotation test which resulted from many experiments including those of James, of Kreidl, and of Wanner. This test stimulates both labyrinths simultaneously. The test in its simplest form may be carried out by placing a patient in a rotating or swivel chair which is turned through ten complete revolutions in about twenty seconds. The patient's eyes should be closed during the turning in order to eliminate ocular nystagmus.

The stimulation of the labyrinth occurs at the beginning and at the end of the turning. For practical purposes it is easier to examine the patient's eyes at the end of the turning. To accentuate the negative acceleration, the chair should be stopped suddenly. An examination is made immediately for the presence of nystagmus, past pointing, falling, head turning, and subjective sensations.

By applying the rules of Flourens, Ewald, and

Arellano, to be described later, it is possible to determine, with any particular head position, which canals have been maximally stimulated, which minimally, and just what nystagmus should normally occur. For instance, when the normal person is turned to the right with head erect there should be horizontal nystagmus to the left which should last for twenty to forty seconds, because the left horizontal semicircular canal has been maximally stimulated.

Post-rotational Nystagmus—Barany, Fischer and Wodak, and Dodge have made careful studies of post rotational nystagmus and have reported that after turning in one direction there may be more than one series of eye movements, each successive series being in opposite direction to the one immediately preceding. They also state that this is true of post rotational sensations. Dussier de Barenne suggests that these successive periods of post rotational nystagmus and sensations are probably not due to anything happening in the labyrinth but are more likely due to "gradually declining, fluctuating after discharges of central processes in the vestibular centers or other parts of the central nervous system." For the above reasons it can be readily seen that timing of post-rotational nystagmus may vary with the individual observer.

Variability in post-rotational nystagmus time has been reported by Van Wulfften Palthe in the human, by Maxwell, Dussier de Barenne, and de Kleyn in the rabbit, and by Borries, and Mowrer in the pigeon. It has been noted that post-rotational nystagmus time may be shortened in persons whose professions involve turning, for example, ballet dancers, skaters, seasoned aviators, and whirling artists. All this suggests the need for caution in attempting to read too much into slight variations which may follow labyrinthine stimulation with any test.

Compensation—Immediately after one labyrinth has been eliminated, for instance by disease or by nerve section, turning to the side opposite the destroyed labyrinth shows a shortening of the post rotational nystagmus time. This time may be reduced to five or eight seconds. However, compensation is soon established and in a matter of weeks or months, after turning to either side there may be little difference in the duration of the nystagmus. This compensation, which occurs when one labyrinth is partially or completely destroyed, impairs the practical value of the rotation test

which stimulates both labyrinths simultaneously. This disadvantage may, however, be overcome by the caloric test, which stimulates each labyrinth separately.

Differential Stimulation of the Individual Vertical Semicircular Canals—The rotation test is of particular value in distinguishing between the reactions of the anterior and posterior vertical canals of one labyrinth. The head may be postured in such a way that the right anterior vertical canal and the left posterior vertical canal can be stimulated simultaneously, or the opposite position of the head may be chosen and the left anterior vertical and the right posterior vertical canals can be stimulated.

THE CALORIC TEST—Kubo, in 1906, demonstrated that the semicircular canals respond to caloric stimulation as well as to electrical and mechanical stimulation.

Barany, in 1907, described a clinical test which consists of syringing one ear with a quantity of fluid above or below body temperature. Barany concluded that the change in temperature caused an up or down movement of the endolymph, i.e., movement in a vertical plane. The endolymph moves relatively to the membranous canal, thereby stimulating the endorgan of any canal in a vertical plane. He found that with the head in one position he could stimulate the horizontal canal, in another position, the vertical canals. It should be noted that in the caloric test, both vertical canals are of necessity stimulated simultaneously.

Kobrak, in 1923, introduced the idea of using a minimal caloric stimulus to test the labyrinth. This improved the test considerably because the patient is less disturbed and more detailed information about the labyrinth can be acquired.

Dohlman, in 1935, experimented upon patients upon whom a radical mastoid operation had been performed and was able to apply caloric stimulation directly to the bony semicircular canals. He found that a change in temperature of one tenth of a degree above or below threshold was sufficient to cause a change in the direction of the nystagmus. He pointed out that in a normal ear, most of the heat or cold of the solution used in the test is expended in changing the temperature of the skin of the external ear, the drum head, the air of the middle ear and the mastoid antrum, et cetera. This means that slight changes in time of onset or duration of postcaloric nystagmus are more

likely to indicate differences in the physical conditions surrounding the bony semicircular canals than changes in the sensitivity of the semicircular canals themselves

When the drum head is perforated and the middle ear dry water should not be used in carrying out the caloric test on that ear because of the possibility of lighting up infection Ethyl chloride may be sprayed into an ear when a perforation is present with less danger of resulting infection However a more agreeable method of carrying out the test is to use Dundas Grant's cold air tube (Fig 277) This consists of a coiled copper tube incased in cloth Air blown through the tube is cooled by the evaporation of ethyl chloride which has been sprayed on the cloth wrapping of the tube Fifteen to thirty seconds of cold air douching through such a tube elicits a reaction from the normal labyrinth

When doing a caloric test in an open mastoid wound sterile hot or cold water may be used However a simple safe and practical method is to spray ethyl chloride into the cavity in the region of the aditus To avoid confusion from any unfamiliar head position immediately after stimulation the head should be turned in such a way that the face is directed to the ceiling The horizontal canal of the tested ear is then in a vertical plane and the nystagmus should be horizontal and to the opposite side

When the caloric test is done under an anesthetic only the slow phase of the nystagmus is elicited The mechanism whereby general anesthesia eliminates the quick phase of nystagmus is not understood

Methods and Equipment—Innumerable modifications of Bárány's and Kobrak's original tests have been described It is important that each clinician choose a simple method for carrying out the test so that he will be able to faithfully reproduce each step in the procedure each time he performs the test For instance the temperature of the water used should be the same for each test An advantage in using ice water is that the temperature is always the same and that only a small quantity is necessary to produce a minimal stimulation Hot water may be used but it is less practical than is cold water However, in the presence of certain types of spontaneous nystagmus the hot caloric test may be a very valuable check on the reactions elicited by the cold caloric test

The methods of introducing the solution vary

greatly One may use a continuous flow from an irrigating can to introduce a measured quantity of fluid or the ear may be irrigated with fluid for a definite period of time The solution may be introduced by a syringe with either the quantity or the time or both being measured The temperature of the fluid should be known

Our aim is to describe a simple labyrinthine test that can be of service to the otolaryngologist in his daily practice If one is carrying out experiments or doing research the caloric test may be as elaborate and detailed as desired

Any hospital ward and every specialist's of



Fig 277—Dundas Grant's cold air tube (half size) A rubber or poltzer bulb or from a compressed air machine is passed through the coil The tube at the small end of the coil at the left of the picture is inserted into the external ear canal through an ear speculum under direct vision

fice contains all the equipment necessary to carry out a satisfactory caloric test An exception to this is a stop watch which is not essential but which is of value A 20 cc Luer syringe and an 18 gauge needle make a very excellent means of introducing the water against the drum head Two to 4 cc of ice water usually elicit a definite nystagmus with slight dizziness and past pointing

Technic and Observations—The patient may be seated but is more comfortable lying down (Figs 278-279) There is an advantage in having the head turned with the tested ear upwards The solution then comes well in contact with the drum membrane The water should be introduced slowly under direct vision care being taken to avoid the presence of an air pocket against the drum head The needle should be well attached to the syringe so that it does not come off and injure the ear The fluid is kept in contact with the drum head for about twenty seconds The ear is then decanted by having the patient turn the head so that the face is directed to the ceiling

Observations should be made when the head is erect and again when the face is directed to the ceiling Nystagmus begins in the normal individual about thirty seconds after the onset of stimulation A record may be kept of the time of introduction of the fluid of the latent

period before nystagmus begins, and the time the nystagmus lasts is erect both vertical canals are being stimulated and the nystagmus will be rotatory In each



Fig 278 —A method of carrying out the caloric test with the patient in a sitting position. Note that the head is well inclined to the side of the nontested ear. The solution is being injected under direct vision by means of a 20-cc Luer syringe and an 18 gauge needle.

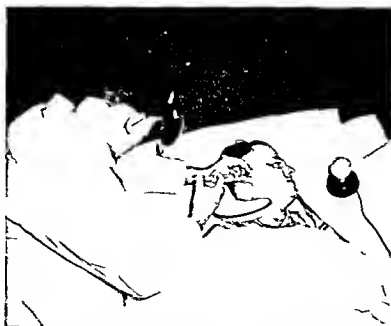


Fig 279 —A method of carrying out the caloric test with the patient lying down. Note that the patient's head is rotated well to the side of the nontested ear. The solution is being injected under direct vision, by means of a 20-cc Luer syringe and an 18 gauge needle.

When the face is directed to the ceiling the horizontal canal is being stimulated and the nystagmus will be horizontal. When the head case the nystagmus is to the opposite side and in the normal individual lasts about one hundred seconds.

The nystagmus which follows stimulation of the horizontal canal is always more marked and of longer duration than that which follows stimulation of the vertical canals. The reason for this has not been adequately explained.

A wide range of variability of postcaloric nystagmus time has frequently been reported in normal individuals. Attempting to attach significance to slight variations makes for confusion and brings discredit on the test.

As soon as the nystagmus has been observed, the patient should be directed to close his eyes. Past pointing of the arms, head turning and,

deduced, it is probably inactive. On the other hand, when the nystagmus lasts longer than two minutes, it suggests that the labyrinth is hyper irritable and to confirm this the test should be repeated using about $\frac{1}{2}$ cc of ice water. When it is found that neither the horizontal canal nor the vertical canals respond to the cold caloric test, valuable use may be made of the hot caloric test which changes the direction of the endolymphatic flow. Further information may be acquired by doing the rotation test with the head postured to stimulate separately the canals in question.



Fig 280—A picture demonstrating past pointing of both arms to the left after stimulation of the left labyrinth with 20 cc of ice water. Note the slight inclination and rotation of the head to the left. There is a slight rotation of the trunk to the left. Note that Figures 280, 281, 282 and 283 all show movements of both arms to the left, together with some turning of the head and body to the left after stimulating the left labyrinth (See text for significance).

falling should then be noted. The patient should be asked to describe his sensations. A note should be made about the amount, the direction, and the character of the dizziness. If there have been spontaneous attacks of dizziness, it is at this juncture that the patient should be asked to compare the sensations which have followed the test with those occurring in the spontaneous attacks.

When the nystagmus time is markedly reduced the test should be repeated with larger quantities of solution. The labyrinth may prove to be hypo irritable if the reaction to 10 cc of ice water is slight. When the labyrinth does not respond to 30 cc of ice water, slowly intro-

duced, it is probably inactive. When there is a spontaneous nystagmus directed to the side opposite the ear being stimulated with ice water, it may be difficult to be sure that the spontaneous nystagmus has been affected by the test, for instance, that a nystagmus to the left has been accentuated by stimulation of the right labyrinth. In such a circumstance the right labyrinth should be stimulated with hot water and if the labyrinth is normal the spontaneous nystagmus to the left should be converted to a nystagmus to the right.

By judicious use of the caloric tests it should be possible to determine whether or not any one semicircular canal is reacting normally.

THE GALVANIC TEST—This test has not been generally adopted clinically. Due to the spread of the electrical current, not only the labyrinth but the eighth nerve and vestibular ganglia are stimulated. For this reason the results of the test are difficult to interpret.

THE PAST POINTING TEST—In the conventional method of carrying out this test, the patient, with closed eyes is asked to move his arms in a vertical or horizontal plane. He is directed to touch a given point of reference usually the examiner's outstretched finger. The labyrinth is then stimulated by the rotation or caloric method and the patient is instructed to

availing misconceptions. They showed conclusively that past pointing is an involuntary limb reflex which is of vestibular origin, just as is eye nystagmus. They further pointed out that by asking the patient to find an external point of reference with the outstretched finger a voluntary corrective factor is needlessly introduced which actually takes away from the clinical value of the test.

Tait pointed out the analogy between a patient (head erect) past pointing to the right after having been turned in a chair ten times to the right and the chair stopped suddenly, and the reaction of a frog which has been



Fig. 281.—The picture of a frog on a turn table turning to the left. This is the way the frog compensates during rotation of the table to the right or after a sudden stop of rotation of the table to the left. The horizontal canal of the right labyrinth is being stimulated. Note the curve of the body to the left, the turning of the head to the left, the backward position of the right leg with the forward position of the left. The right arm is relatively forward.

move his arms in the same plane as before and to touch the point of reference (Fig. 280). If both the patient's arms move to the same side of the point of reference (i.e., if the patient "past points") the reaction is an index of semi-circular canal response. It is a true compensatory reaction and will be in the same direction as the slow phase of the nystagmus. Nystagmus indicates the intactness of certain intracranial pathways and the arm movements indicate the intactness of totally different intracranial and spinal pathways.

Dorcus and Mowrer reviewed the phenomenon of past pointing and cleared up many pre-

turned rapidly to the right and the turning suddenly stopped. The patient and the frog both tend to turn to the right after the turning ceases because in each case the negative acceleration has stimulated the left horizontal semicircular canal. Tait further suggested that if the aforementioned patient were supported on his hands and knees in the position of a frog (Fig. 281), the movements of his arms to the right and the bending of the upper part of his body to the right (Fig. 282) would be the exact reaction necessary to begin turning to the right on all fours. He also pointed out that this would explain the "disk throwers

reaction' described by Fischer and Wodak (Fig 283)

When a lesion of the labyrinth causes a spontaneous past pointing of the arms, both arms are usually affected in the same sense, whereas if the lesion causing the past pointing is in the central nervous system the homolateral limb is more likely to be affected. Grahe discussed this influence of the central nervous system on past pointing. In order to make any dissociation more obvious he advocated simultaneous movement of both arms.

The following rule may be applied when judging the results of the past pointing test

the membranous labyrinth there will be spontaneous attacks of dizziness. The fistula test should always be carried out in the presence of chronic suppurative otitis media, particularly when the patient has complained of dizziness.

The test consists of compressing the air in the external ear canal and this is easily done by using a Siegle's speculum which will fit the canal tightly. When a fistula is present, the membranous labyrinth, if still intact, will be stimulated by the change in air pressure. This elicits an immediate, violent, momentary nystagmoid movement of the eyes and a sensation of dizziness. The direction of the nystagmus



Fig 282



Fig 283

Fig 282 —The picture of a man on hands and knees on a turn table turning to the left to compensate during rotation of the table to left or after a sudden stop of rotation of the table to the right. The horizontal canal of the right labyrinth is being stimulated. Note the curve of the body to the left, the inclination of the head to the left, the relative forward and to the left position of the right arm with the left arm being correspondingly back and to the left.

Fig 283 —The disk thrower's position. This picture was taken after the left labyrinth had been severely stimulated with cold water. The man was asked to stand with eyes closed and arms outstretched. After a short latent period the arms, body, and head slowly assumed the positions shown in the picture. An illustration of extreme past pointing. (From Fischer, M. H. and Wodak, E. *Arch f d ges Physiol* 207 1924.)

past pointing, head turning and falling after labyrinthine stimulation are always in the opposite direction to the quick phase of the nystagmus.

THE FISTULA TEST —This test consists of applying a moderate amount of mechanical pressure to the membranous labyrinth through a fistula in the bony labyrinthine wall. The site of the fistula is usually in the horizontal canal.

In a normal ear this test would be negative. When there is chronic suppuration in the middle ear with defective drainage the bony labyrinthine capsule is frequently eroded to cause a fistula. If the disease stimulates or irritates

depends on the position of the fistula and upon whether the air in the external ear canal has been compressed or aspirated. The direction is of no great clinical significance. The important fact is that if the test is positive, we have learned that there is a fistula in the labyrinthine capsule, that the membranous labyrinth is still able to react to stimulation, and that the internal ear is not as yet extensively invaded.

When the test is negative in a case of chronic suppurative otitis media and there is a history of dizziness, it is still possible that a fistula is present and that the labyrinth has been invaded and destroyed. In such a case the exact condi-

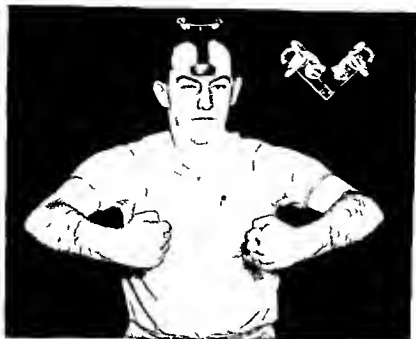


Fig 284 —Both arms are held approximately in the plane of the horizontal canals the fists representing the ampullae and the bowed arms the canals proper. Note that the ampullae represented by the fists are at the forward ends of the canals. On the demonstrator's head is a Negus model of the labyrinth which in the clinic helps visualization of the semicircular canals. Inset is a picture of Arellano's models described in Figure 276.



Fig 285 —The model is holding his arms so that the left arm represents the left anterior vertical canal and the right arm the right posterior vertical canal. These canals are in the same diagonal plane as is clearly demonstrated in Figure 276. Note that the ampulla is at the lower end of each canal but that each is in the same relative position to the other as are the ampullae of the horizontal canals shown in Figure 284 and the ampullae of the opposite pair of vertical canals shown in Figure 286. Inset is a picture of Arellano's models described in Figure 276.

tion of the internal ear should be determined by means of the caloric, rotation, and hearing tests. This should be done immediately because of the danger of the infection spreading from the internal ear to the meninges. When the fistula test is positive complete and free external drainage of the mastoid and middle ear should be established at once in order to protect the inner ear from suppurative involvement.

In the modern operation of fistulization of the labyrinth as a treatment for deafness, most of the patients react positively to this test. The history will readily differentiate these patients

analyzed, can be used to determine the normal reactions which should follow various test procedures:

1. Stimulation of a semicircular canal elicits nystagmus in the plane of that canal. (Flourens)

2 A horizontal semicircular canal is maximally stimulated by a movement of the endolymph in the canal towards its ampulla. A vertical semicircular canal is maximally stimulated by a movement of the endolymph in the canal away from the ampulla. (Ewald)

3. When a semicircular canal, either horizontal or vertical, is maximally stimulated it elicits a nystagmus (quick phase) to its own side. Minimal stimulation causes a nystagmus to the opposite side. (Ewald)



Fig. 286.—The model is holding his arms so that the right arm represents the right anterior vertical canal and the left arm the left posterior vertical canal. Note that the fists represent the ampullae and are at the lower ends of the canals. Inset is a picture of Arellano's models described in Figure 276

from those suffering from active middle ear infection.

Interpreting Labyrinthine Reactions—As one carries out the labyrinthine tests one should try to visualize what is happening in the individual canals and to deduce therefrom what eye and arm reactions should normally follow certain forms of stimulation. Figures 284, 285, and 286 have been included in order to simplify this visualization process and to help the student to determine the relation of any one canal to any particular head and body position.

The following condensed rules, if carefully

4. Nystagmus due to rotation is always in a plane at right angles to the axis of rotation, irrespective of the position of the head. (Arellano)

5 Post-rotational nystagmus (quick phase) is always in the opposite direction to the rotation. (Arellano)

Variations from normal in reactions from labyrinthine stimulation are significant only after nonlabyrinthine factors have been assessed. Extralabyrinthine factors, such as the thickness of the drum, the presence of a perforation in the drum, or the presence of an aural polyp in the external canal, may alter the results of the caloric test.

Hypo- or hyper- sensitivity of the labyrinth may be due to a variety of causes, such as increased labyrinthine pressure, intoxication, vascular changes, anemia, or Meniere's syndrome

Complete absence of the vestibular responses on one or both sides is most likely due to destruction of the labyrinth or eighth nerve and is frequently, but not necessarily, associated with a hearing loss. When the onset is sudden it suggests a vascular lesion.

Loss of response from only the horizontal canal on the one hand or only the vertical canals on the other, whether on one or both sides, suggests a lesion in the posterior fossa. When associated with a hearing loss or a facial weakness it strongly suggests an eighth nerve lesion on the same side.

Either perversion of nystagmus or dissociation of eye movements suggests a brain-stem lesion.

A spontaneous vertical nystagmus indicates a brain stem lesion probably located near the midline.

Loss of conjugate deviation or loss of the quick phase of the nystagmus may be due either to a cortical or a brain stem lesion.

Marked unilateral deafness indicates that the lesion is in, or peripheral to, the cochlear nuclei. A lesion central to the cochlear nuclei does not cause marked unilateral deafness because of the symmetrical bilateral cortical representation from the cochlea.

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MÉNIÈRE'S SYNDROME

There is still much controversy as to what constitutes a case of Meniere's syndrome Under this heading are placed most of the cases of deafness, tinnitus, and dizziness occurring in association with nonsuppurative disease of the labyrinth without evidence of organic disease in the central nervous system The term "Meniere's disease" is not generally used because the pathologic basis is not known The original idea of there being a hemorrhage into the labyrinth has been abandoned

A patient suffering from a typical attack gives a history of sudden episodes of dizziness associated with tinnitus and deafness The three symptoms, however, may not occur simultaneously, one symptom preceding the others by months or years, in which case the term "atypical Meniere's syndrome" is generally used

Pathology.—Our knowledge of the pathologic background of Ménière's syndrome is meager It is possible that if all the facts were known this syndrome represents several different pathologic conditions Mygind and Dederent (1932) advanced the hypothesis that Ménière's syndrome is due to a "water logging" of the labyrinth

It was as late as 1938 that Hallpike and Cairns published the first report of a histologic study of the labyrinth in two patients who were

known to have had the symptoms of Ménière's syndrome shortly before death. They have published subsequent confirmatory reports and sensory elements of the internal ear. They suggested that owing to the presence of changes in the region of the saccus endolymphaticus,



Fig 287—Normal right cochlea (From Hallpike C S, and Cairns H. Observations on the Pathology of Ménière's Syndrome. *J Laryng & Otol* 53)

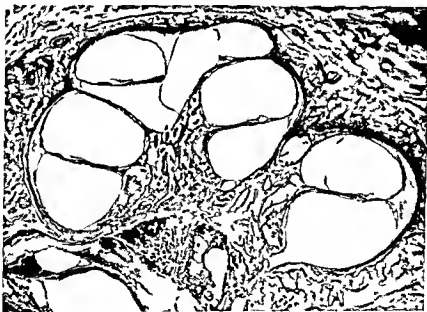


Fig 288—The left cochlea. Gross dilatation of the scala media has occurred with displacement of Reissner's membrane on to the wall of the scala vestibuli. At the apex Reissner's membrane has been displaced through the helicotrema into the scala tympani (From Hallpike C S, and Cairns H. Observations on the Pathology of Ménière's Syndrome. *J Laryng & Otol* 53)

their findings were corroborated by Lindsay in 1942. Hallpike and Cairns reported gross distention of the endolymphatic system (Figs 287, 288) together with degenerative changes in the

there may be a failure in the endolymph absorbing mechanism. Lindsay referred to the condition as 'labyrinthine dropsy' (Figs 289, 290)

The possibility of Meniere's syndrome being an allergic phenomenon has received much consideration. In the experience of the authors,

cent of their cases and in none did the symptoms seem to be attributable to an allergic sensitivity.

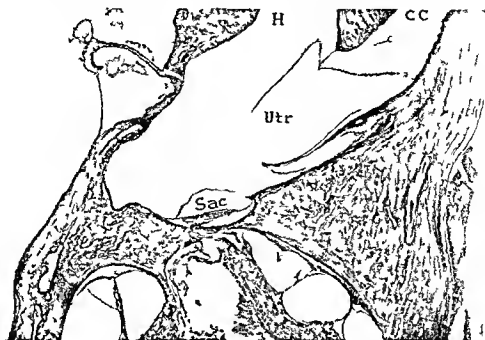


Fig 289 —Section of a normal labyrinth in approximately the same plane as that of Figure 290 showing the saccule (Sac) the utricle (Utr) the opening of the horizontal canal (H) and the opening of the common crus (CC) (Courtesy of J R Lindsay)

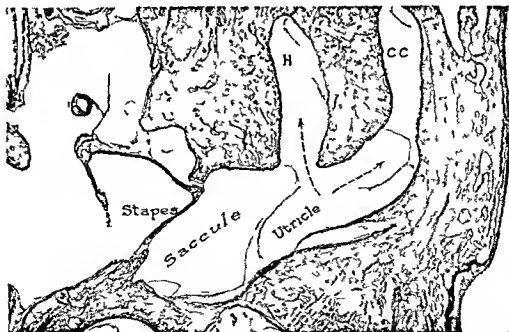


Fig 290 —Dilated saccule with the wall in contact with the foot plate of the stapes. Herniations of the utricle into the perilymphatic space of the small end of the horizontal canal (H) and the opening of the common crus (CC) are denoted by arrows (Courtesy of J R Lindsay)

who have reported the study of over 100 cases, a personal or family history of allergy could not be ascertained in more than 25 per

Symptoms and Diagnosis.—*Vertigo*, which Brain so aptly defines as 'the consciousness of disordered orientation of the body in space,'

is the most disturbing symptom in Meniere's syndrome. In the classical case, the attacks of dizziness occur suddenly, often when the patient is at rest and even when he is asleep. The patient feels that he is whirling or that objects about him are whirling. The attack may last for only a few minutes, it may be severe for weeks, or it may persist in a subacute form for months. The dizziness is not necessarily a rotatory or whirling sensation. As a rule the patient's sensations may be reproduced by stimulating the labyrinth as in the caloric test. When the symptoms are reproduced it is very suggestive that the spontaneous dizziness is due to labyrinthine irritation. It is therefore important to have the patient compare the dizziness of his attacks with the sensations he experiences immediately following the caloric test.

To prove that labyrinthine stimulation may cause sensations other than the classical whirling dizziness, the authors recorded the subjective sensations of a number of patients immediately after their labyrinths had been stimulated by the caloric test. Some felt that they were going to faint, others felt unsteady, others experienced a rocking sensation, a staggering sensation, a swimming sensation, a sensation of weakness, a sensation of backward swaying or a wavy sensation. Our conception of Meniere's syndrome should, therefore, be broad enough to include any one of these complaints as a possible substitute for rotatory dizziness.

Crowe stated that in his series of cases of Meniere's syndrome the caloric test was normal in 35 per cent, subnormal in 19 per cent, and that in 29 per cent the labyrinth of the affected side failed to react. He also stated that in some of the patients in whom there were no reactions to the test at one time, there was a subnormal reaction at a later time. He interpreted this as indicating that there is fluctuation in the pathologic process within the labyrinth. From the foregoing it is readily seen that the results of the caloric test are not diagnostic of Meniere's syndrome.

When the labyrinth does not react to the caloric test, care should be taken to exclude organic disease of the central nervous system, such as an eighth nerve tumor, before a diagnosis of Meniere's syndrome is made. The authors have elsewhere reported a case in which the patient had the classical signs of Meniere's syndrome for two years before definite evidence of intracranial disease occurred.

At operation an eighth nerve tumor was removed.

The authors found that vertigo or dizziness was a complaint in 10 per cent of 600 consecutive admissions to the Montreal Neurological Institute. In the records of the Royal Victoria Hospital dizziness occurred in twenty-one of fifty cases of hypertension in cardiovascular disease, in ten of twenty-three cases of hypotension, in two of twenty cases of traumatic shock, in eighteen of ninety cases of gallbladder disease, in eight of fifty cases of diabetes mellitus, and in six of twenty-five cases diagnosed as avitaminosis. It is possible that in these cases the sensation described as dizziness may not have been reproduced by the caloric test. The authors have seen a number of so-called Meniere's syndrome cases occurring in association with or during the convalescent period following organic heart disease.

Dizziness may be present in the course of acute or chronic middle ear suppuration. Its presence then suggests the imminent danger of involvement of the internal ear by the suppurative process.

Nystagmus may or may not be present during an attack of Meniere's syndrome.

Deafness in Meniere's syndrome usually affects the high tones and is in the nature of a nerve deafness. The grade of deafness is not diagnostic but frequently may be very severe and progressive. The deafness may affect both ears in different degrees or may be confined to one ear.

Usually associated with the dizziness and deafness is *innitus*. This is generally present in the deaf ear and in the ear with the greater amount of labyrinthine irritation. The cause of the tinnitus is not completely understood. It is presumed to arise within the cochlea but it has persisted in some cases after the eighth nerve has been sectioned. There is no characteristic pattern to the tinnitus.

When the tinnitus is unilateral it is helpful in deciding which of the two ears is more likely to be responsible for the vertiginous attacks. Some observers have reported that during a typical attack of Meniere's syndrome tinnitus is exaggerated. This has not been our experience.

Headache and loss of consciousness are not typical of Meniere's syndrome. They do occur in some patients but they are more typical of organic disease in the central nervous system.

favor it whenever the subject harbors a condition that combines two dominant factors—(1) factor X which lies in the sex chromosome and (2) a factor A which lies in one of the autosomes. It is suggested that the latter modifies the mesenchyma reaction, especially that of the osteoblasts and osteoclasts. Sex-linked genes act differently as between the sexes, and may thus possibly affect calcium metabolism.¹ Deficiencies in the mesenchyma occasionally occur in other places than in the otic capsule, leading to exostoses, brittle bones, and blue sclerotics.

The fissula ante fenestram, which is the connective tissue communication between the vestibule and the middle ear in the area between the stapes and the cochlea, is resistant to ossification, but contains, in some fetuses and young children, areas of cartilage. Such areas later change to atypical or unstable osseous tissue which gradually obliterates it with new formed bone resembling that seen in otosclerotic bone.² However, otosclerosis does not originate in the fissula. The fissula is thought to be possibly a seepage or drainage channel for the perilymph. If this is so, a surgically established fistula (or fissula) might serve as an artificial seepage channel for perilymph drainage. All this is hypothetical.

Clinically and histologically the incidence of otosclerosis in the population appears to be quite different, which as Guild³ has pointed out, invalidates most contentions as to the hereditary factors. His latest statistics taken from 1161 routine autopsies show the incidence of otosclerosis in children under five years of age to be less than 0.6 per cent. In persons over five years of age it was approximately 4 per cent, distributed as follows:

Group	Total Number in Group	Number Who Had Otosclerosis	Proportion with Otosclerosis	
			Per centage	Approximate Rate
White females	163	20	12.3	1 in 8
White males	355	23	6.3	1 in 15
Negro males	347	4	1.1	1 in 87
Negro females	135	1	0.7	1 in 135
All whites	518	43	8.3	1 in 12
All Negroes	482	5	1.0	1 in 96

The percentage (4 per cent) should be higher if the sex and racial proportions of those coming to autopsy had been similar to those in the general population. Only one-half of the persons with otosclerosis showed active lesions at autopsy. The incidence in activity was greatest under twenty, and diminished rapidly after sixty years of age.

Exciting Factors.—Otosclerosis may occur at any time of life, but most frequently is manifest near puberty, during pregnancy, the puerperium, and the menopause. It appears (clinically) in individuals usually otherwise healthy, and may be with or without an hereditary taint. It may occur with syphilis, ozena, nervous diseases, rickets, anemia, hypertension, hypotension, arteriosclerosis, or, in fact, with any other of the ills of human flesh.

Studies of identical twins with otosclerosis often disclose an earlier date of onset and a more severe degree of deafness in the twin giving a history of more head colds or hypertrophied lymphoid tissue in the nasopharynx.⁴ Studies in blood chemistry in twins give no convincing clues as to the cause of otosclerosis. They follow remarkably similar patterns in each pair, but since the clinical diagnosis has always been made long after the onset of the lesions the laboratory work also has necessarily been done long after the onset.

Theories as to etiologic factor or factors have been proposed by Wittmaack, O. Mayer, Habermann, Bruhl, Siebenmann, Gray, Guggenheim, and many others, but none has been generally accepted.⁵

Pathology and Histology.—Otosclerosis is a primary affection of the labyrinthine capsule, in the majority of instances bilateral, but not always symmetrical. It resembles in some respects the lesions found in the group of so-called osteodystrophies that affect other bones including the temporal, especially the lesions in von Recklinghausen's and Paget's disease. The new-formed bone is sharply defined and has some of the characteristics of immature bone (Figs 291-298). The islands of cartilage characteristic of the normal enchondral layer of the capsule bone are absent. The lesion starts in one or more foci (even when diffuse), and possesses a distinct tendency to progress slowly. The point or points of predilection are usually located immediately anterior to the oval window and less frequently at the round window near the insertion of the membrana tympani secundaria.

the best available medical treatments should be thoroughly exhausted before surgical treatment is undertaken. Even though the surgical treatment of Meniere's syndrome is more definite than the medical treatment, it should be noted that, as a rule, surgery eliminates the offending organ and does not repair it. Treatment is always unsatisfactory if the organ removed is necessary to the patient's well being. Before submitting to surgery the patient's disability from the disease should be sufficiently great that he is prepared, if necessary, to sacrifice part of his hearing and part of his balancing mechanism to obtain relief.

NERVE SECTION.—The most suitable cases for surgical treatment are those in which the condition is obviously limited to one ear (Dandy). If there is a possibility of both ears being involved the two vestibular nerves can be sectioned while the cochlear nerves are left intact. The results of the bilateral operation may be disturbing to the patient.

The most common operation is to sever one entire eighth nerve or in some cases only the vestibular division of one nerve.

OBLITERATION OF THE LABYRINTH.—Other operations recommended but less frequently carried out have as their aim the destruction of the offending labyrinth by labyrinthectomy or by alcohol injection.

It is to be hoped that when the pathologic basis of Meniere's syndrome is better understood the treatment will be more adequate.

Prognosis.—In any one case it is very difficult to estimate the prognosis. In many cases the condition is progressive. As already stated, remissions are a common feature and may explain many of the reported cures. Some patients apparently recover from one attack and are afterwards free from symptoms. There are, however, cases in which the patient is completely disabled. In many of these, surgical treatment is required before relief is obtained.

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OTOSCLEROSIS*

Otosclerosis (otospongiosis) is primarily a disease or disorder of the bony wall (capsule) of the labyrinth. It is often called "progressive deafness" but is not synonymous with this term unless it actively involves the stapes and annular ligament.

Etiology.—Predisposing Factors.—Otosclerosis is due to a reaction of the otic capsule to one or more as yet undetermined causes. Heredity is an important factor in most instances, as it is in many other tissue dyscrasias. Davenport's studies,¹ as well as those of others, show that clinically in the white race twice as many females as males are affected. In Negroes the incidence is lower and the ratio between the sexes is less, suggesting a susceptibility dependent in part upon evolutionary factors. Other types of obstructive deafness affect the races and sexes more equally.

When both parents are otosclerotic all, or nearly all, of their daughters are otosclerotic, or suffer some type of deafness, whereas only half to two thirds of the sons take on the disease. The least objectionable hypothesis is otosclerosis develops under environments that

*The author gratefully acknowledges the guidance of Dr. Franz Altmann in the selection of photomicrographs and in the description of bone pathology of otosclerosis.

Twenty two ears had 2 otosclerotic areas each, and 5 ears had 3 areas each. The total number of otosclerotic areas in the 81 ears is 113

rior or the inferior margin, or along both margins, of the oval window. These extensions are not counted as separate areas.



Fig 293 —Peripheral part of an otosclerotic focus composed of red bone (Silver impregnation of the fibrillae after Bielschowsky-Maresch). C, Old capsule. O, Otosclerotic focus. (From the histological collection of E. P. Fowler, Jr., College of Physicians and Surgeons, Columbia University.)



Fig 294 —Otosclerosis, red bone. M.P., Mosaic pattern. M.S., Marrow spaces with wide vessels. (From the histological collection of the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University.)

"Otosclerosis was present anterior to the oval window in 65 of the 81 ears. Most of these areas had extensions backward along the supe-

* The round window region is next to the oval window region in number of otosclerotic areas. Twenty five of the 81 ears had an otosclerotic

█ Guild's latest statistics³ for eighty one ears whom sections of both ears are available, (forty nine patients) with otosclerosis in 1161 "32 had bilateral otosclerosis, 14 had unilateral



Fig 291—Otosclerotic focus blue bone Lacunar erosion of the old labyrinthine capsule BB Blue bone O Osteoclastic destruction of the blue bone OC Osteoclastic erosion of the old capsule (From the histological collection of E P Fowler Jr College of Physicians and Surgeons Columbia University)

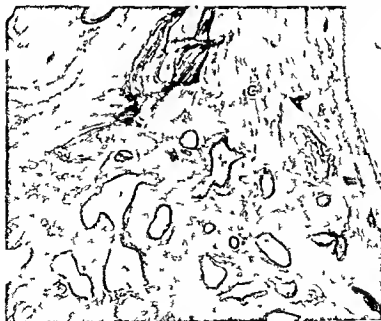


Fig 292—Peripheral part of an otosclerotic focus composed of red bone (Hematoxylin-eosin specimen.) C, Old capsule O Otosclerotic focus (From the histological collection of E P Fowler, Jr., College of Physicians and Surgeons, Columbia University)

routine autopsies reveal the location of the otosclerotic areas in forty six of the patients in

Nine of the 14 patients with unilateral otosclerosis were white women, 3 were white men

Twenty-two ears had 2 otosclerotic areas each, and 5 ears had 3 areas each. The total number of otosclerotic areas in the 81 ears is 113.

rior or the inferior margin, or along both margins, of the oval window. These extensions are not counted as separate areas.



Fig. 293.—Peripheral part of an otosclerotic focus, composed of red bone (Silver impregnation of the fibrillae after Bielschowsky-Maresch.) C, Old capsule, O, Otosclerotic focus. (From the histological collection of E. P. Fowler, Jr., College of Physicians and Surgeons, Columbia University.)



Fig. 294.—Otosclerosis; red bone. M.P., Mosaic pattern. M.S., Marrow spaces with wide vessels. (From the histological collection of the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University.)

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*Guild's latest statistics*³ for eighty-one ears whom sections of both ears are available; (forty-nine patients) with otosclerosis in 1161 "32 had bilateral otosclerosis, 14 had unilateral,



Fig. 291.—Otosclerotic focus; blue bone. Lacunar erosion of the old labyrinthine capsule. B.B., Blue bone. O., Osteoclastic destruction of the blue bone. O.C., Osteoclastic erosion of the old capsule. (From the histological collection of E. P. Fowler, Jr., College of Physicians and Surgeons, Columbia University.)



Fig. 292.—Peripheral part of an otosclerotic focus, composed of red bone. (Hematoxylin-eosin specimen.) C, Old capsule. O., Osteoclastic focus. (From the histological collection of E. P. Fowler, Jr., College of Physicians and Surgeons, Columbia University.)

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Fig 294 —Otosclerotic red bone MP Mosaic pattern MS Marrow spaces with wide vessels (From the histological collection of the Department of Otolaryngology College of Physicians and Surgeons Columbia University)

Otosclerosis was present anterior to the oval window in 65 of the 81 ears. Most of these areas had extensions backward along the supe-

The round window region is next to the oval window region in number of otosclerotic areas. Twenty five of the 81 ears had an otosclerotic

area at some part of the attachment of the round window membrane usually it was along continuous with the area anterior to the oval window



Fig. 295 —Blue mantles (B M) (From the histological collection of the Department of Otolaryngology College of Physicians and Surgeons Columbia University)

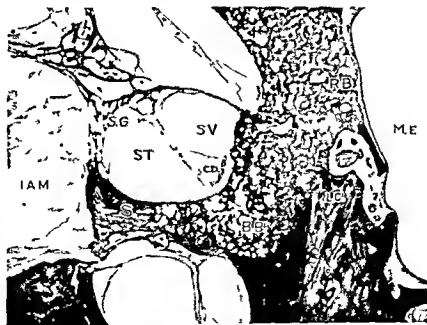


Fig. 296 —Large otosclerotic focus in the lateral wall of the cochlea extending into the septum between the basal and middle turn BB Blue bone CD Cochlear duct with organ of Corti IAM, Internal auditory meatus. LC Nonaffected part of the labyrinthine capsule ME Middle ear RB Red bone S Septum between basal and middle turn SG Spiral ganglion ST Scala tympani SV Scala vestibuli

the lateral part of the attachment, sometimes also anteriorly or posteriorly. Only in 3 of the 25 cases was the area near the round window

The number of otosclerotic areas in each of the other locations in which they occur in this material is

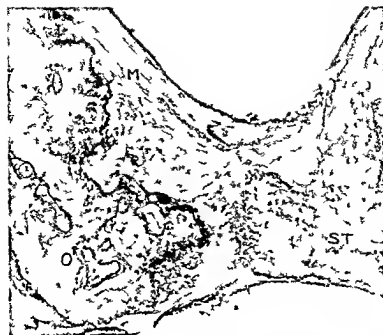


Fig 297—Otosclerotic focus in the oval window without stapes ankylosis. M Mucosa of the middle ear. O Otosclerotic focus. ST Stapes. (From the histological collection of the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University.)

* Stapedial footplate (primary areas only not including cases of ankylosis)	10
* Anterior part of cochlear capsule	6
* Inferior part of cochlear capsule (not reaching internal auditory canal)	5
* Anterior inferior part of cochlear capsule	1
* Fundus of internal auditory canal (inferior part)	2
* Superior part of cochlear capsule	1
* Cochleariform process (limited to this structure only)	1

In size the otosclerotic areas in this material vary from ones less than a millimeter in the longer axis to one that had replaced the entire cochlear capsule except for part of the anterior region and the modiolus and had extended into the oval and the round window regions.

The lining membrane of the middle ear (mucoperiosteum) usually shows little or no change from normal. However, otosclerosis may and often does occur in ears showing evidence of past nonsuppurative or suppurative otitis media.

Under these conditions it is apparently not a sequela of the middle ear condition but merely coincidental. Otosclerosis is too severe a lesion to heal (cease) without leaving pathologic changes in the tissues involved, and there is no evidence that it ever recedes or disappears.

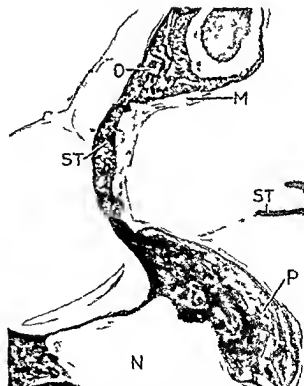


Fig 298—Otosclerosis at and in the oval window with stapes ankylosis and involvement of the foot plate. M Mucosa of the middle ear. O Otosclerotic focus in capsule. ST Stapes with focus in foot plate. P Promontory. N Niche of round window. (Photomicrograph by courtesy of S. R. Guild.)

Otosclerotic foci have been observed in every layer of the (human) capsule. When active, the lesion consists of slow progressive changes which may be briefly described as follows:

Localized resorption (absorption) of the original bone of the capsule by lacunar erosion.

Replacement of the absorbed bone by immature weblike bone containing much cementum and few fibrils (so-called 'blue bone') (Fig 291) (Nager and Mayer believe this scarcity in fibrils causes it to stain darkly blue with hematoxylin-eosin).

Repeated lacunar resorption and replacement by later generations of weblike bone which gradually becomes more and more mature, therefore richer in fibrils and poorer in cementum, hence taking on a reddish stain (so-called 'red bone') (Figs 292, 293).

This red bone is sooner or later, in turn and to varying extents, replaced by lamellar bone, which also stains red in hematoxylin-eosin.

The continuation of the process results in the formation of a brecciated bone with irregular patterns of mosaic design (Fig 294).

Depending upon the distribution and size and patency of the blood vessels in the marrow spaces the otosclerotic areas may take on sclerotic, vascular, medullary, cystic, or fibrous growth types.

The otosclerotic process may become stationary at any period of its development and may start up anew in previously apparently quiescent areas. It may be inactive in some and very active in other areas.

The growth of the foci occurs either by lacunar erosion on a broad front or by means of finger-like projections of immature bluish-staining bone surrounding blood vessels located in the center of these projections (the blue mantles of Manasse) (Fig 295). Blue mantles are also found without any connection with the otosclerotic foci and are believed by Nager⁶ to represent the preliminary stages of the lesion.

The extent of the lesion varies from minute and inactive areas to extensive and active involvement of the whole capsule, including the semicircular canals and both the labyrinth windows. The bony growth may penetrate into the basal coil of the cochlea with the production of fibrous tissue and new bone in the perilymphatic spaces. As far as is known, otosclerosis is the only disease of the human labyrinthine capsule which acts in exactly this way.

When the bony growth encroaches upon the internal auditory meatus the eighth nerve may be compressed. This is extremely rare. Studies in bone pathology have elicited much as to the histogenesis of otosclerosis but nothing definite as to its origin or treatment.

Symptoms.—There are no general symptoms. The local are subjective and otologic. The chief

symptom is a disturbance in the hearing, depending in part upon the amount of obstruction in the oval and round windows, and in the acoustic labyrinth spaces, interfering with normal functioning of the auditory neural elements.

Progressive loss of hearing, at first more severe in the lower octaves of the audible frequency scale, and without other apparent cause, is the most characteristic symptom. The deafness is not at first noticeable, or is slight, and it may be months or years before it cannot be ignored. It may, therefore, appear to be precipitated by psychosomatic upsets, mental or bodily overexertion, pregnancy, childbirths, or overindulgence in alcohol, tobacco, or drugs, all of which may depress the hearing irrespective of the presence of otosclerosis. Coincidental middle ear or other inflammatory episodes may add their share to the deafness already present.

The typical pure obstructive loss is encountered more often in the young. Characteristic air-conduction audiograms coincidental with the first definite diagnosis are shown in Figure 299. These graphs are not necessarily those of otosclerotics. Similar losses of hearing may be found in association with other ear disorders and diseases acquired during the life of the individual.

Tinnitus may be a most intense, troublesome, and frequent symptom accompanying otosclerosis. It is seldom entirely absent except in very young patients. It is often described as a buzzing or rumbling noise, but also appears as a high-pitched tone or whistle which may be present for variable periods of time. Tinnitus usually increases with the deafness, and vice versa. It may continue even with total deafness. The loudness of the tinnitus, even when apparently unhearable, is usually equivalent to a sound not over 5 to 10 decibels over the threshold, and is usually easily masked by environmental noise. Acoustic hyperesthesia is not infrequently encountered and usually paracusis wilisiana is present. The ability to hear best in a noisy place may be explained by the fact that the hearing for speech appears better only because the obstructive deafness excludes much environmental noise, and the speaker's voice is automatically raised in intensity to overcome the masking effects of the noise.

In some patients lapses of memory, depression, dizziness, neurasthenia, and feelings of heaviness and tightness in the head are encountered. Psychologic reactions differ greatly—

some patients almost ignore the annoying symptoms and others become obsessed and tortured by them. They are worse in neurotic, anemic, and poorly nourished people. Thyroid and other endocrinopathies frequently obscure the picture.

Diagnosis—There is no sure diagnostic sign for clinical otosclerosis. Other forms of progressive deafness may be clinically undistinguishable from it. The only sure diagnosis is that made at autopsy, but here the lesions found do not tell us how much the hearing had been affected. The common criteria for the diagnosis of clinical otosclerosis are a positive family his-

ter, a diminished external auditory canal reflex, and moderate hyperostoses in the external auditory meatus. There is also tendency toward a diminished movability of the malleus. If the hearing formerly has been very acute (5 or 10 decibels above average normal), a 15 to 20 decibel loss may appear of little consequence on the audiogram, but it is a definite and important loss and is often a symptom of an impending or potential deafness.

Otosclerosis can apparently occur by itself or be complicated by any of the lesions to which the ear is subject, but it is rarely seen at autopsy in chronic suppurative ears. In its latest stages,

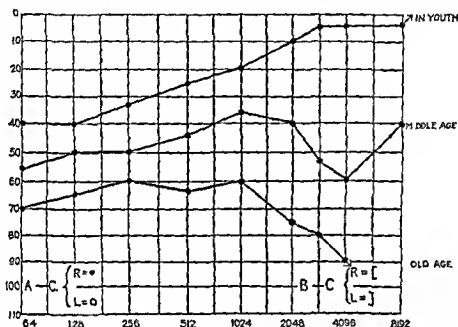


Fig. 299—Typical audiograms in otosclerosis. In youth bone conduction is usually near normal at all frequencies. In middle age bone conduction is often below normal for some of the frequencies above 1024. In old age bone conduction is usually below normal in the upper half of the frequency scale. Whether otosclerosis is present or not. Similar audiograms occur with other forms of deafness. In otosclerosis, however, at all ages the low tones are always lowered no matter what the loss may be in the higher tones.

tory, a progressive deafness (in its early stages more marked in the lower octaves of the frequency scale) without apparent cause, patent eustachian tubes, normal or so-called 'increased' bone conduction, and usually a normal drum membrane—inconstantly a pinkish glow from the promontory shows through the membrane. In the aged the drum is often dull, lusterless, and atrophic. (The drum membrane is best examined with the aid of a magnifying pneumatic otoscope, a daylight bulb being used for illumination.) There is a tendency to changes similar to those often found following local irritation (such as are noted in swimmers). These changes include a diminished quantity of

particularly in persons past middle life, it is almost always accompanied by involvement of the neural mechanism of hearing as indicated usually by losses in the upper half of the frequency scale by both air and bone conduction, and by the appearance of the recruitment phenomenon.⁷ Thus nerve deafness usually does not differ from that found in the population at large at similar ages who have no sign of otosclerosis, but it is not inconceivable that the lesion itself may not somehow also have involved the neural apparatus of hearing.

Diagnostic Tests—All of the old tests are of little aid when compared to a careful audiometric test made in a properly sound proofed room.

with the aid of a standard audiometer at threshold, and also above threshold, to bring out the presence or absence of the recruitment phenomenon,⁷ the latter being the only sure sign of involvement of the neural mechanism of hearing

In persons with otosclerosis the Gelle test often gives positive findings but is uncertain. In the Weber test the sound is usually referred to the more obstructed side of the head. The Schwabach test shows normal or increased bone conduction and diminished air conduction. The reaction to the Rinne test is usually negative.

In the absence of nerve deafness, bone conduction is usually average or high normal for the low and middle tones (speech tones), in young persons it is usually average or high normal for all tones. It may be determined for the lower tones with the aid of a tuning fork or the bone conduction receiver of the audiometer, for the tones above 512, the bone conduction receiver is more dependable.

The threshold of hearing is uncertain,⁸ especially for the low tones. It frequently varies (up and down) as much as 10 decibels (and sometimes 15 decibels) at one testing. Small changes in intensity of a tone (2 or 3 decibels) may not be sensed, or are sensed with difficulty. In nerve deafness the opposite is true, as small changes are easily sensed. There is often a persistence of the tone sensation after the sound has ceased ("after image" or "after sound").

Complications and Sequelae.—These are due to the increase in size of the growth and the more extensive involvement of the annular ligament, the stapes and the round window membrane, the labyrinth spaces or the internal auditory canal. The disease is never fatal, so that most histopathologic material has been acquired from routine autopsies.

Treatment.—There is no cure for the lesions of otosclerosis. Even its prevention or arrest will not change its inheritability under favorable environmental conditions.⁵ Prophylactic treatment may consist in the prevention of conception or birth of the child, either by prohibiting the marriage of otosclerotics, by sterilization, by the use of contraceptives, or by the termination of pregnancy, or similar radical methods. Prenatal preventive treatment in otosclerotic families at present consists in careful supervision of the mother during pregnancy and the puerperium. Blood chemistry determinations and endocrine checkups should be

made if possible before pregnancy, at its beginning, and at two month intervals during pregnancy and lactation. A balanced diet, with ample vitamin and mineral intake, should be maintained. Even during lactation the baby as well as the mother should have careful medical supervision. The child should have otologic examinations, especially six months prior to and once a year after puberty. In addition to the otologic examination, calcium, phosphorus and phosphatase determinations as well as the determination of mineral intake and the basal metabolic rate should be made. Correction of all abnormalities should be attempted. Hypertrophied lymphoid tissue in the nasopharynx should be watched, and if excessive or diseased should be removed by operation, and subsequently irradiated if it shows a tendency to return. The tonsils should be removed only when greatly hypertrophied or when diseased. Unwarranted removal may increase rather than decrease the tendency to nasopharyngeal lymphoid recurrence.

When there is any tendency to ear disease, lip reading should be taught early. Social, educational, and occupational rehabilitation is important when deafness occurs and here the leagues for the hard of hearing have been of inestimable service to both the patient and the otologist. The well to do, as well as the poor, can obtain much help by and through these agencies.

Hearing aids (air conduction or bone conduction, approved by the A.M.A.) are efficacious in improving the hearing in persons deafened from otosclerosis. The bone conduction receiver is usually preferred because bone conduction is good, and the receiver is hidden behind the auricle, the external meatus remaining open and free from the irritation and annoyance of the air conduction ear piece. However, some otosclerotics prefer air-conduction hearing aids because these are more efficient.

Operative Treatment.—No present operative treatment is curative as far as the otosclerotic process is concerned, but this does not necessarily mean that the loss of hearing due to superimposed causes can never be improved nor that operative measures can never improve substantially the deafness due to the otosclerosis. Artificial drum membranes and plugs of various substances, or of tissue (*i.e.*, Hughson's round window graft), are of questionable value in otosclerosis. Several decompression oper-

ations have been experimented with, namely, exposure of the sacculus endolymphaticus, spinal and suboccipital punctures, and fenestration of any or all of the semicircular canals or vestibule

The pioneers in the fenestration operation were Passow, Barány, Jenkins, Holmgren, and Sourdis. Many modifications in technics have been practiced and discarded by these experimenters. Lempert and his followers have, during the past five years, revived interest in the procedure and encouraged further experimentation. To a competent and properly trained operator there is not much difficulty in learning the technic, but many months of training on the cadaver should precede operations on the live subject.

The great obstacle to the success of the fenestration operation has been the tendency of the fistula to become obliterated by formation of new bone. It is evident from the reports of the men doing the greater number of these operations, that no sure method of preventing this closure has yet been devised. In other words, no one can tell the patient that even if his hearing is improved immediately following the operation (which it usually is) that it will remain improved for so many months or so many years following the operation. If the fistula does close, or if the hearing for any reason goes down even slightly after what may appear to the patient to be a satisfactory improvement, the result is still often tragic. The mental reaction may be even worse than when the hearing was originally lost. In this connection one must not forget that no matter how much the hearing for the speech frequencies is improved on the basis of the lowering of the threshold, that unless the threshold is near or above the 30 decibel loss line the hearing will not be what is called 'satisfactory' even for near speech. The patient wants satisfactory hearing, not just temporary satisfaction, and not just some improvement in hearing.

The commonly accepted indications for the fenestration operation are (1) Obstructive deafness from otosclerosis in properly selected cases in which there is inability or compelling aversion to wearing a hearing aid. (In almost every person with obstructive deafness a properly selected and fitted hearing aid is capable of improving the hearing distance to a greater extent than can be expected through the fistula operation.) (2) An obstructive deafness of 35

decibels or more in the frequency area most useful for speech (which when bilateral is equivalent to at least a 27 per cent over all loss of hearing for speech—Fowler's table⁹) associated with relatively little nerve deafness.

There have been a few reports of spectacular improvements even though air conduction and bone conduction were both at very low levels. This suggests that both the labyrinth windows had been affected by the otosclerosis. If the recruitment phenomenon is absent in the presence of severe loss of bone conduction, it is an indication that the deafness is either total or not of nerve origin. In other words, the bone-conduction loss is then not necessarily a true measure of nerve deafness.

The contraindications for the fenestration operation are (1) Moderately severe or severe nerve deafness, particularly in the frequency areas most useful for speech. (2) Recurrent or chronic suppurating ears. (3) Adhesions immobilizing the movement of the drum membranes. (Note: This may not be a contraindication in some instances.) (4) Osteitis deformans (Paget's), osteitis fibrosa (von Recklinghausen's disease), osteogenesis imperfecta (fragilitas ossium), or any of the endocrinopathies in which the labyrinthine capsule is attacked.

The operation being a major procedure, the patient should be a good surgical risk. One reason for the low mortality rate has been the care taken in this direction.

The diagnosis of otosclerosis is so uncertain that without doubt many of the fistula operations have been performed on patients with obstructive deafness due to causes other than otosclerosis. It seems possible that more satisfactory results may be obtainable in these wrongly diagnosed cases.

Although the fundamentals for the fenestration operation have not been altered through the years, many changes in technics and devices have developed in the endeavor to find some way to prevent the closure of the operative fistulas. Fistulas have been made in the semicircular canals, their ampullae, in the vestibule, or in several locations. Various cutting, grinding and cleansing procedures, tissue and other grafts, and obturators, have been devised to diminish trauma or to prevent or discourage the closing of the openings by bone regeneration. All are foreign bodies, and therefore irritative and incite of connective tissue and bone-scar formation in varying degrees.

The latest modifications in technic, devised by Lempert, are given in the article that follows

Animal experimentation has as yet failed to solve the problem, but some progress is being made. For details the reader is referred to the contributions of those who have been most interested in this experimental surgery 5 10 11

Operative hazards include injuries to the fallopian canal and facial nerve, to the membranous labyrinth, and the semicircular canals or the vestibular membranes, with consequent inflammatory reactions and loss of function of the labyrinth

Paralysis of the facial nerve and prolonged vertigo are postoperative complications which, in the great majority of instances, clear up. Increased or total deafness occurring postoperatively does not clear up. Infection of the wound and delayed healing often require many months and even years of postoperative care. Closure of the fistula, excessive scabs, and granulations require repeated operations

A person who has had a fenestration operation should be forbidden to swim or use water in the ear. Cold winds or draughts and violent blowing of the nose should be avoided

The operation is of little hazard to life. Less than 1 per cent of those operated upon have been reported to have died from the operation

Although the operation apparently has had no curative effect upon the lesion, yet in the great majority of instances there has been an immediate improvement in the hearing. Overall statistics of functional results are very misleading because some surgeons report over 80 per cent of those operated upon with a satisfactory level of hearing (30 decibels and less below normal) and others report only 30 per cent (one to five years after the operation). If the hearing is below the 30-decibel level the value of the operation is questionable in view of the risks involved. The hearing level obtained soon after the operation is apparently seldom if ever maintained, but in spite of this or of no improvement, some patients derive from the operation a sense of satisfaction and a better adjustment to their handicap. They feel that they have at least done all that can be done and there is no urge for further struggling to bring back the hearing

EDMUND PRINCE FOWLER

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TECHNIC OF THE LEMPert FENESTRATION OPERATION

On the basis of my observations and findings following 900 fenestration operations for clinical otosclerosis, I feel that it is necessary, as a preface to any description of my surgical procedure, to state concisely (1) the surgical principle upon which this surgery is based, (2) the objectives to be aimed for as a result of this surgery, and (3) the present-day indications for this procedure, realizing that our conception of the fundamentals is rapidly changing

Surgical Principle.—Though the origin of and the cure for the pathologic lesion of otosclerosis have both remained unknown, middle ear deafness resulting from and accompanying such a lesion is today curable by surgical means. By ignoring the otosclerotic tumor in the region of the oval window which ankylosed the stapedial footplate and thus impedes the transmission of air-borne sound to the organ of Corti, and surgically creating a new oval window in the surgical dome of the vestibule where the otosclerotic lesion was never observed to have

existed, the perilymph and endolymph are rendered mobile once more for air-borne sound and hearing is thus improved

Objectives—These embrace (1) the improvement of hearing to the level necessary for social and economic rehabilitation and (2) the continued maintenance of the improved hearing

Present-Day Indications—The fenestration operation is indicated in the deafened (1) When the existence of stapedial footplate ankylosis, due to otosclerosis, is suspected and the diagnosis of clinical otosclerosis can be made with reasonable certainty (2) Where there is reason to suspect that there still exists in the ear chosen for operation a reservoir of cochlear nerve func-

The entire auricular region is covered with a sterile gauze dressing, which is held in place with a two-inch bandage

Anesthesia—A combination of analgesia and local anesthesia is employed *Analgesia* (as outlined by Dr J Branower) for the average adult 3 grains of neonal and $4\frac{1}{2}$ grains of nembutal given by mouth, two and a half hours prior to the scheduled time of operation This is to be followed by $\frac{1}{4}$ grain of morphine sulfate given hypodermically forty five minutes later This is followed thirty minutes later by a 3-grain nembutal suppository, to be followed forty five minutes later by another hypodermic injection of $\frac{1}{4}$ grain of morphine sulfate. The

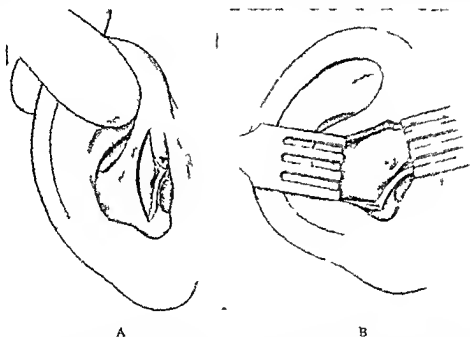


Fig 300—A Incisions B Endaural antiauricular exposure of the mastoid portion of the temporal bone

tion in excess of what is being tapped by the functionally impeded air conduction mechanism, and that this reservoir is sufficient to permit the improvement of hearing as a result of reconstructing the air conduction mechanism (3) Where there does not exist at the time of operation an active middle ear infection, whether acute, subacute, or chronic The tympanic membrane must be intact and not perforated

Technic.—Preparation of Surgical Field—The night before operation, a one-inch area of hair is shaved in the region of the auricle The auricle and surrounding skin and the external auditory canal are cleansed with green soap and water, washed with alcohol, and then tincture merthiolate 1 1000 is instilled into the cana

patient is moved to operating room thirty minutes later *Local anesthesia* is induced by a 1 per cent solution of procaine hydrochloride and 1 20,000 solution of epinephrine hydrochloride injected into the skin, fibrous tissue, and periosteum of the antiauricular suprameatal membranous triangle and the membranous portion of the posterior wall of the external auditory canal

Surgical Technic—1 CREATION OF THE END AURAL ANTIAURICULAR EXPOSURE OF THE MASTOID PORTION OF THE TEMPORAL BONE—Three endaural incisions are made The first is begun in the membranous lining of the superoposterior wall of the external auditory canal, at the junction of its osseous and membranous por-

tions This incision is carried downward and outward along the entire membranous portion of the posterior wall of the canal until the lower end of the anterior border of the concha is reached The second is begun at the point of commencement of the first incision in the superoposterior wall of the canal, is carried along and through the membranous lining of the outer third of the superoposterior wall, and is continued upward adjacent to the tragus, into and along the anterior wall of the antauricular suprameatal membranous triangle, through the skin, the fibrous tissue, and the periosteum up to the apex of this triangle The third, which connects the first two incisions along the an

With a periosteal elevator the entire triangular flap is subperiosteally lifted from its attachment to the bony surface The upper and lower ends of this flap are then freed from their final attachments with the aid of curved scissors This triangular flap, which consists of the outer third of the membranous lining of the posterior and the superoposterior walls of the canal and contains hair follicles and ceruminous glands, is removed and discarded The removal of this membranous flap results in an endaural extra cartilaginous membranous window for the antauricular surgical approach to the temporal bone This endaural membranous window is now mobilized by a subperiosteal elevation of

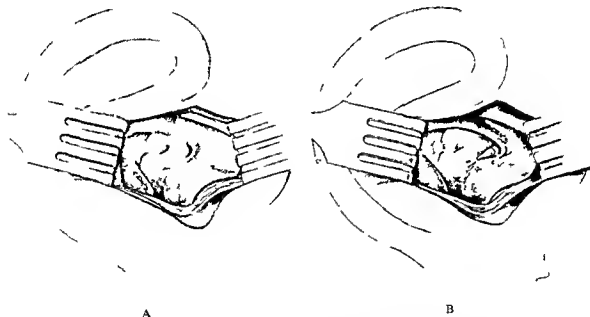


Fig. 301—A Exposure of the mastoid aspect of the labyrinthine base B Transaural opening of the eptympanum and exposure of the incudomalleal joint

tenor border of the concha is begun from the outer end of the first incision at the lower end of the anterior border of the concha is carried upward through the skin, the fibrous tissue, and the periosteum along the entire anterior border of the concha, and is continued along and adjacent to the anterior border of the helix which forms the posterior boundary of the antauricular suprameatal membranous triangle, up to the apex of the triangle where it meets the outer end of the second incision These incisions are so designed that they remain extra cartilaginous and extramuscular A triangular flap of membranous tissue results from these three incisions

both the postauricular skin and periosteum covering the outer mastoid cortex and the antauricular skin and periosteum covering the posterior root of the zygoma Two retractors are inserted and held in position by an assistant

With the retractors in situ, the endaural membranous window can be moved in any desired direction over the temporal bone to permit the necessary surgical intervention

2 EXPOSURE OF THE MASTOID ASPECT OF THE LABYRINTHINE BASE.—With an electrically-driven serrated dental bur placed on the outer mastoid cortex in the region posterior and immediately adjacent to the spine of Henle and

its course guided inward in a line parallel with the superoposterior bony wall of the external auditory canal, the mastoid antrum is entered. The opening in the outer cortex leading to the floor of the antrum is widened with round, sharp, electrically-driven cutting burs and the rest of the cortex is gradually removed, exposing the entire mastoid cell structure. Employing the antrum floor as our point of orientation, all the supra-antral, pensinus, and retrofacial cell structure is exenterated with burs and sharp curets, until the entire inner bony table of the mastoid process covering the middle and posterior fossae and the lateral sinus is exposed to view. The perilabyrinthine cell structure on the

thus exposing to view the mastoid course of the horizontal portion of the fallopian canal and the epitympanum with the incudomalleolar joint and the anterior malleolar ligament.

4 RETROTYMPANIC OPENING OF TYMPANUM AND EXPOSURE OF THE INCUDOSTAPELIAL JOINT, AND CHORDA TYMPANI NERVE—The entire cutaneous lining of the skeletonized posterior, superoposterior, and superior bony walls of the external auditory canal is slowly and gently separated from these walls a few millimeters at a time with a specially devised narrow periosteal elevator and the correspondingly freed portions of the bony walls are removed with a specially devised narrow rongeur biting forceps. The

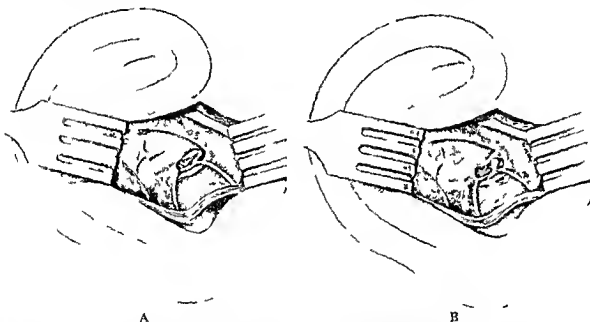


Fig 302 —A, Retrotymppanic opening of tympanum and exposure of the incudostapedial joint and chorda tympani nerve. B Exposure of surgical dome of vestibule for fenestration. Removal of incus.

floor of the antrum and antral portion of the aditus is carefully removed with sharp curets until the entire mastoid aspect of the base of the labyrinth is exposed to view with the external, superior, and posterior semicircular canals sharply defined.

3 TRANSANTRAL OPENING OF EPITYMPANUM AND EXPOSURE OF THE INCUDOMALLEAL JOINT—With electrically-driven, round, sharp, cutting burs, the outer bony zygomatic cortex is removed, exposing the zygomatic cell structure. Employing sharp curets, the cellular structure covering the mastoid aspect of the posterior, the antral aspect of the superoposterior, and the epitympanic aspect of the superior bony walls of the external auditory canal is removed,

sulcus tympanicus on either side of the notch of Rivinus corresponding to the thus removed bony canal walls is slowly and carefully skeletonized and removed away from the fibrocartilaginous corresponding margins of the tympanic membrane. The above described surgical maneuvers must be executed without disturbing the continuity of the outer dermal layer which is the only layer binding and uniting the tympanic membrane with the cutaneous lining of the external auditory canal. Employing a sharp curet, part of the pyramidal eminence is also removed.

We have now exposed to view the long process of the incus, the incudostapedial joint, and the chorda tympani nerve.

5 EXPOSURE OF THE SURGICAL DOME OF THE VESTIBULE FOR FENESTRATION—(a) Removal of the Incus—The articular capsule of the incudomalleal joint is divided, and the body of the incus is separated from the head of the malleus. The long process of the incus is then disconnected from the stapes, and the incus is removed and discarded. This exposes to view the amputated ends of the external and the superior semicircular canals, the oval window with the stapes in situ within the tympanic wall of the vestibule, and the tympanic transverse portion of the facial canal lying external and superior to the oval window and separating it from the

of the vestibule, is seen the epitympanic antero-most portion of the facial canal as far as the geniculate ganglion

6 CREATION OF THE FENESTRA NOV-OVALIS IN THE SURGICAL DOME OF THE VESTIBULAR LABYRINTH—The vestibular labyrinth consists of an osseous labyrinth, with a perilymphatic membranous labyrinth occupying its lumen and an endolymphatic membranous labyrinth suspended within the perilymphatic labyrinth. Because of the minute and extremely delicate anatomic structures dealt with in the process of creating the vestibular fenestra, the technic is best divided into several distinct stages

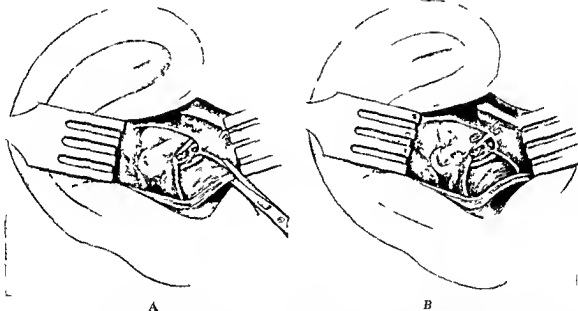


Fig. 303—A, Exposure of surgical dome of vestibule for fenestration. Removal of head and neck of the malleus. B, Creation of the fenestra nov-ovalis in the surgical dome of the vestibule

dome of the vestibule. Exposed to view is also the niche for the fenestra rotunda, as well as the posterior aspect of the cochlear promontory. In removing the incus, care must be exercised not to injure the chorda tympani nerve.

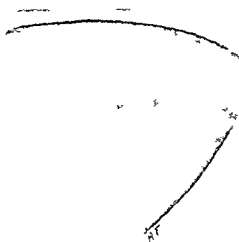
(b) Removal of the Head and Neck of the Malleus—With a specially devised malleus clipper, the head and neck of the malleus is amputated in the region immediately superior to the attachment of the anterior malleolar ligament. This exposes to view the surgical roof of the vestibule and the processus cochleariformis with the tendon of the tensor tympani muscle attached to the manubrium mallei. External and superior to the processus cochleariformis, separating this structure from the roof

Stage 1—With the aid of Zeiss magnifying glasses, an area about 5 mm in length and 2 mm in width of the osseous labyrinth in the region of the surgical dome of the vestibule is slowly and carefully worn down with a dental plug finishing bur as far as its inner endosteal bony layer. This innermost bony layer, when extremely thinned out, becomes transparent and assumes a bluish gray appearance because it reflects the lumen of the vestibule underneath it.

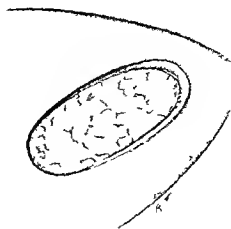
Stage 2—The thinned out, bluish-gray, transparent area of the endosteal layer of the osseous labyrinth is gently and slowly fractured and pulverized with the same dental plug finishing bur. The fractured and pulverized innermost layer of the osseous labyrinth is now resting on

the endosteal membrane lining the lumen of the vestibule which surrounds the perilymph and forms the perilymphatic membranous labyrinth

Stage 4—Employing a small rubber-bulb syringe with warm physiological solution of sodium chloride, the presenting surface of the



Stage 1

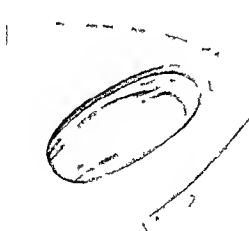


Stage 2

Fig. 304—Fenestration of vestibular dome Stages 1 and 2

Stage 3—With a specially devised microscopically pointed needle, all the fractured, decimated, and pulverized particles of the endosteal bony layer of the osseous labyrinth are carefully removed from the somewhat depressed

endosteum in the fenestrated area of the osseous labyrinth is gently irrigated until severed and washed away. The membranous perilymphatic labyrinth is thus opened and the membranous endolymphatic labyrinth is exposed to view



Stage 3



Stage 4

Fig. 305—Fenestration of vestibular dome Stages 3 and 4

surface of the endosteum, taking extreme precaution not to injure and to leave completely intact the perilymphatic membranous labyrinth. Fenestration of the dome of the vestibulo-osseous labyrinth is now complete

Whereas formerly all these four stages of technic were employed to create the fenestra nov-ovalis, in the last hundred cases I omitted the fourth stage of this technic. I left the endosteum lining the lumen of the vestibule intact

and did not open the perilymphatic labyrinth. This was done in the belief that the membranous endolymphatic labyrinth could thus be protected against undergoing severe postoperative inflammation which sometimes results in a protracted period of vertigo and occasional damage to the organ of Corti. The end results obtained when fenestration was accomplished in this manner would seem to justify this modification of technic, whenever achievement is technically possible.

7. CREATION OF THE PLASTIC TYMPANOMEATAL FLAP AND COVERING THE FENESTRA NOV-OVALIS WITH SHRAPNELL'S MEMBRANE — One cone-shaped incision, the apex of which is

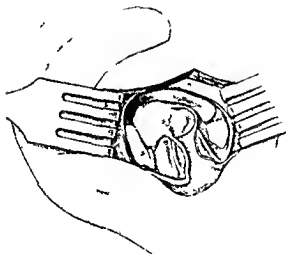


Fig. 306 — Plastic tympanomeatal membrane in position and skin grafted mastoid wound

the posterosuperior margin of the tympanic ring and the base is the outer margin of the superior bony wall of the external auditory canal, is made through the cutaneous membrane lining the superior bony wall of the external auditory canal. Another cone-shaped incision is made through the cutaneous lining of the inferior bony wall of the external auditory canal, the apex of which is the postero inferior margin of the tympanic ring and the base is the outer margin of the inferior bony wall of the external auditory canal.

Following these lines of incision, one elevates subperiosteally the cutaneous lining covering the bony roof and floor of the canal down to the tympanic ring, where only its outer

dermal layer joins and becomes continuous with the outer dermal layer of the tympanic membrane. The drum is then freed from the entire superior margin and the posterior half of the inferior margin of the sulcus tympanicus and thus is converted into a movable and replaceable membrane. The cone-shaped meatal portion of the tympanomeatal membrane is now unfolded and is gently pulled backward into the mastoid wound. In being pulled backward, it carries with it the mobilized tympanic membrane. The tympanomeatal membrane is so placed that the tympanic portion thereof seals the entire tympanic air space, with Shrapnell's membrane covering the entire fenestra nov-ovalis, and the periosteum lined meatal portion of the tympanomeatal membrane covers most of the floor of the mastoid wound.

8. SKIN GRAFTING AND SEALING OF POST-OPERATIVE MASTOID WOUND — To keep the degree of postoperative inflammation at a minimum and to shorten the healing period, the inner bony table of the postoperative mastoid wound is covered with very thin Thiersch skin grafts obtained from the hairless skin of the patient's thigh. The entire postoperative cavity and the external auditory canal is filled with liquid paraffin up to the margin of the outer mastoid cortex.

9. POSTOPERATIVE CARE OF PATIENT — Upon returning to his room the patient is given intravenous injection of 1000 cc of 5 per cent glucose and saline, and an ampule of coramine hypodermically.

10. POSTOPERATIVE CARE OF WOUND — Constantly employing the strictest aseptic precautions, the superficial mastoid dressing is changed daily. Five days postoperatively the first deep dressing is performed, when the solidified paraffin mold is removed in one piece and the wound is painted with a 2 per cent aqueous solution of gentian violet. The postoperative wound is cleansed thereafter every other day. Healing is usually complete within a period of five to six weeks.

End Results Obtainable — 1. Fenestration of the labyrinth in clinical otosclerosis can result in the restoration of practical, serviceable hearing for all social and economic contacts, and such improved hearing can remain continuously when the newly-created vestibular fenestra remains open. M. E., a housewife, has retained her hearing improvement for the past six and a half years.

2. Fenestration of the labyrinth can restore to normal the hearing in a totally air-conduc-

obtained and retained such a hearing improvement for the last two years

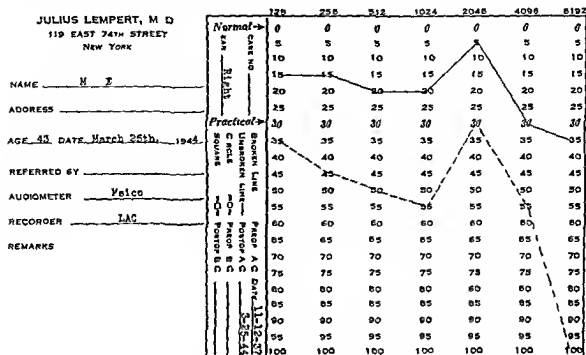


Fig 307—Audiogram of a patient in whom practical hearing was restored. The hearing has been maintained for the last six and a half years

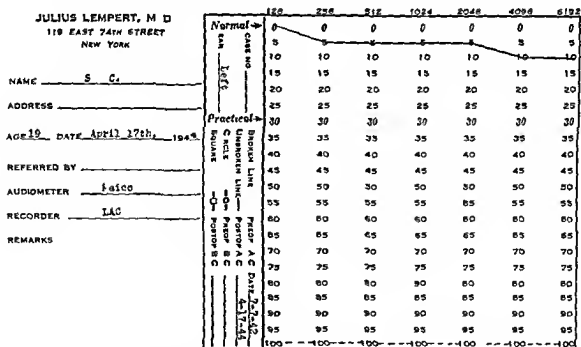


Fig 308—Audiogram of a patient in whom normal hearing was restored and continuously maintained in a totally deafened ear

tion deafened ear and such normal hearing can be continuously retained S C., a schoolboy,

3 Following the fenestration operation the continued progress of deafness, which is char-

acteristic of clinical otosclerosis, can be arrested in the operated ear, while in the non-operated ear the hearing loss continues its progressive downward course. This has been observed in most of the patients in whom one ear was successfully fenestrated. In M E, the

aids us in analyzing the pathogenesis of congenital diseases of bone.

The bone of the endochondral capsule is the only bone in the human being that normally persists throughout life without change. Its limited internal reconstruction comes to an end

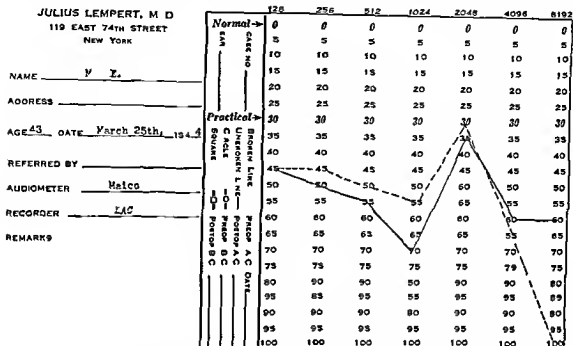


Fig 309—Audiogram Broken line = hearing in left unoperated, ear November 12, 1937 Unbroken line = hearing in left, unoperated ear, March 25 1933 The hearing in the unoperated ear had grown progressively worse

housewife, who had her right ear operated upon in November, 1937, the hearing in nonoperated left ear has become progressively worse, while hearing in the operated right ear has remained at highest postoperative level (See Figs 303, 305)

JULIUS LEMPERT

LABYRINTHINE CAPSULE LESIONS WITH GENERALIZED DISEASES OF THE SKELETON, ENDOCRINOPATHIES, AND METABOLIC DISEASES

GENERALIZED DISEASES OF THE SKELETON

Generalized diseases of the skeleton should be of interest to the otologist (1) because of possible bearing on otosclerosis and (2) because the unique structure of the labyrinthine capsule

by the end of the second year of life and its structure remains stabilized throughout life in the form in which it originally developed. It therefore maintains many primitive (embryonic) features, i.e., skeinlike bone ("Strahlenknochen" of the Germans), globuli ossei, and rests of calcified cartilage. The periosteal layer shows internal reconstruction throughout life as does the rest of the skeleton. These facts help to determine at which period of embryonic life certain congenital diseases of the skeleton appear.

Osteoporosis.—Atrophic or osteoporotic processes occur in senility or may be caused by any chronic disease which leads to cachexia, general debility, or prolonged decubitus (an insufficient diet, achloridia, anemia, et cetera).¹ The hearing threshold is usually not lowered. Roentgenograms of the spine show demineralization, especially in the bodies of the vertebrae, which may finally collapse. The blood calcium, phosphorus, and phosphatase are normal. Phosphatase is an enzyme which hydrolyzes the

phosphoric esters of the blood. The blood relies on this enzyme for the replacement of its calcium and phosphorus. When absorbed into the blood there is an increased secretion of calcium in the urine (calculi) as in osteoporosis.

In senility there is a tendency to osteoporosis but this, if it involves the petrous bone, attacks the periosteal layer whereas the endochondral capsule remains unaffected for a long time.

The changes are caused by a disturbance of the process of physiologic internal reconstruction which goes on in the skeleton throughout life. There is normal resorption of the bone tissue but reduced new formation of bone. As a

anemia with anisocytosis and poikilocytosis. Roentgen-ray examination shows a marked generalized osteosclerosis with narrowing or obliteration of the central marrow cavity of the long bones, the bone presents a homogenous, marble-like appearance.

The essential feature of the bone changes is a disturbance in the growth of the bone. Of the two factors which bring about the physiologic internal reconstruction of bone, one, the osteoclastic destruction of the old bone, is reduced, whereas the other, the apposition of newly formed bone, takes place in a regular or moderately increased degree. (The process is the re-

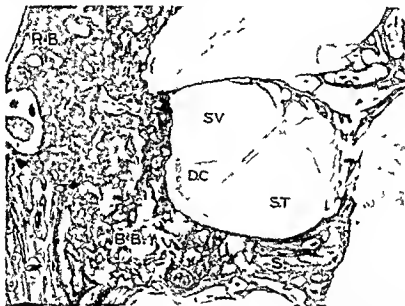


Fig. 310.—This photomicrograph, showing the lesions of otosclerosis is inserted here to facilitate comparisons with the other uncolored photomicrographs of lesions of the temporal bone. It is identical with the kodachrome shown in Figure 296, page 374. B.B., Blue bone. D.C., Ductus cochlearis middle turn. R.B., Red bone. S., Septum between lower basal and middle turn. S.T., Scala tympani. S.V., Scala vestibuli. (From the histological collection of the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University.)

result the marrow spaces become large and the trabeculae thin. In the labyrinthine capsule the periosteal layer is chiefly involved whereas the endochondral layer remains for a long time intact.

Albers-Schönberg Disease (Marble Bones).—Osteosclerosis is a generalized familial disease of the skeleton with recessive hereditary traits. It becomes manifest almost invariably during the growth period. Clinically it is characterized by a marked tendency to bone fractures but with good prospects for healing. Caries of the teeth are noted, and very frequently there is atrophy of the optic nerve. In the majority of cases, there is a hypochromic

verse of that observed in osteoporosis.) In the endochondral bones this abnormality becomes manifest even during the primary ossification. The transformation of the primary into the secondary marrow spaces occurs more slowly than usual. The secondary or definitive marrow spaces are smaller and the bony trabeculae which border the marrow spaces are thicker than normal, owing to the prevalence of the deposition of bone over its resorption.

There is increased formation of membranous bone, and the internal reconstruction of this bone is reduced. The weblike fibril bone persists almost in toto and new lamellar bone is deposited on this weblike fibril bone. Thus the

marrow spaces are gradually and progressively narrowed. The changes in the labyrinthine capsule are the result of these processes.

The endosteal layer of the labyrinthine capsule is thickened; the endochondral layer contains more globuli ossei than normal; the periosteal layer is thickened and contains only a few small marrow spaces. The inner ear seems to be intact and the hearing unaffected.²

Osteitis Fibrosa Cystica (von Recklinghausen's Disease, Parathyroid Osteitis).—The parathyroids control and regulate the calcium and phosphorous metabolism and when abnormal lead to disease of the bones. Parathyroid tumors cause osteitis fibrosa cystica with its hyperparathyroid syndrome. The bones become poor in calcium and soft as a result of osteoporosis. The

spaces being filled with fibrous and hemorrhagic tissue. The endochondral layer may be affected late in the process if at all. The endosteal layer and the inner ear remain unaffected. There is no deafness unless it is caused by other lesions. The changes in the otic capsule in so-called renal osteitis fibrosa cystica (renal rickets) are identical with those in von Recklinghausen's disease.

Osteitis Deformans (Paget's Disease).—In osteitis deformans or Paget's disease there are no changes in the parathyroids and the calcium phosphorus balance is undisturbed. Phosphatase activity is increased. The disease is considered to be a localized monosteal or polyosteal affection and not a generalized disease of the skeleton. In the labyrinth capsule the changes



Fig. 311.—Labyrinthine capsule in Albers-Schönberg disease in an infant thirteen months of age. A.L., Apposition line; G.O., Globuli ossei; E.C., Endosteal capsule; L.B., Lamellar bone; P.C., Periosteal capsule; S.B., Skein like bone; I.L.B., Incompletely lamellar bone.

loss of calcium takes place by lacunar erosion in the marrow spaces and the latter become fibrous and hyperemic; the normal honey trabeculae being replaced by a network of weblike fibril bone (*geflechtartiger Knochen*). Cyst formation within the bone marrow, hemorrhages and the so-called giant cell or brown tumors are pathognomonic. These latter contain many multinucleated giant cells with hemosideratic pigment in the protoplasm. The formation may histologically resemble giant cell sarcoma but are regarded by many authors as a kind of inflammatory reaction of the tissues to the hemorrhages.

The labyrinthine capsule changes consist in diffuse bone resorption by lacunar erosion throughout the periosteal layer; the marrow

start in the periosteum and gradually affect all the layers. In advanced cases only small remnants of the old capsule are preserved. The newly formed bone shows a similar structure to that observed in von Recklinghausen's disease but cysts and brown tumors are absent. The cementum lines in the compact areas of newly formed bone represent late stages of the disease and form an irregular network. This is known as the mosaic pattern of Schmorl and is characteristic of Paget's disease.

In some cases the inner ear contains a serous exudate which possibly explains the nerve deafness and dizziness not infrequently encountered. In others the latter is due to deformation of the nerve canals by the newly formed bone with stretching of the nerve fibers. In other cases the

conduction deafness may be due to bone and calcium deposits in the stapediovestibular joint

Rickets and Osteomalacia.—These diseases are due to disturbances in calcium metabolism. Rickets occur in children, and osteomalacia in adults and old persons. The skull bones, not being subjected to much weight-bearing, suffer less distortion than the weight bearing bones of the skeleton, but the bony capsule shows typical changes confined to the periosteal layer (with newly-formed bone lamellae). In the marrow spaces the newly-formed-bone lamellae are poorly calcified and show a red-eosinophilic (red stained) osteoid border which is typical for

It is caused by dysfunction of the osteoblasts, possibly on the basis of a vitium primae formationis of the entire mesenchyma, the osteoblasts producing insufficient and structurally abnormal bone tissue. The physiologic osteoclastic destruction of the endochondral bones shows normal extent, but according to some authors it may be increased. In the affected bone the cortex is in this way made very thin, in many places interrupted, and the trabeculae of the spongiosa are scarce and insufficiently developed. Spontaneous fractures are common.³

The disease occurs in two forms, as osteogenesis imperfecta congenita and as osteo-



Fig. 312.—Paget's disease, vertical section through the temporal bone. C.C., Remnants of the old cochlear capsule. C.T., Cavum tympani. I.A.M., Internal auditory meatus. M.T.T., Tensor tympani muscle. N.B., Newly-formed bone. N.VII., Facial nerve. (Photomicrograph from Otological Research Laboratory, College of Physicians and Surgeons, Columbia University.)

these two diseases. In children the marrow is lymphoid, in old people it is fatty. The endochondral layer remains intact unless active bone resorption occurs. The inner ear remains normal. In ricketic children there is an inclination to suppurative otitis media and tardy healing, but with these exceptions, unless the labyrinthine windows are affected, the clinical significance of rickets and osteomalacia is not of prime importance to the otologist.

Osteogenesis Imperfecta (Fragilitas Ossium).—This is a generalized disease of the skeleton. It manifests itself in a disturbance of the endochondral as well as of the periosteal ossifications

genesis imperfecta tarda? (osteopsathyrosis). The congenital form is a disease of the developing skeleton, the juvenile and adult types a disease of the growing or completed skeleton respectively.

In the labyrinthine capsule the congenital form shows quantitative and qualitative disturbances of ossification. There is a reduction in the amount of bone formation; this is not marked in the endosteal but is marked in the endochondral and periosteal layers. The marrow spaces are very wide, the trabeculae sparse and thin. The skeinlike bone ("Strachinenknochen") of the endochondral layer is struc-

turally abnormal. The number of the osteocytes is increased and there is an appearance as though more osteoblasts than normal have been

in the labyrinthine capsule. The rest of the temporal bone shows similar but less marked changes than does the skeleton as a whole.



Fig. 313.—Endochondral capsule in osteogenesis imperfecta in a newborn child. GO, Globulus osseus; IS, Interglobular space; ASB, Atypical skeinlike bone.

required to produce a smaller amount of skeinlike bone. Similar but less obvious changes have been observed by some authors in the bone of

Osteogenesis imperfecta tarda is frequently associated with blue sclerae and otosclerotic foci in the temporal bones. This triad is called

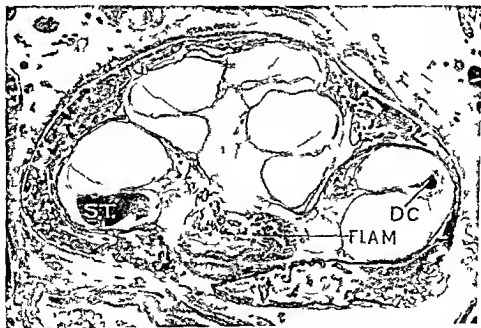


Fig. 314.—Cochlear capsule in osteogenesis imperfecta in a stillborn child (eighth lunar month). DC, Ductus cochlearis filled with fresh blood; ST, Scala tympani filled with fresh blood; FLAM, Fundus of internal auditory meatus filled with fresh blood, resulting from a transverse fracture of the labyrinthine capsule intrapartum.

the periosteal layer. The inner ear structures appear to be normal.

The late form does not show definite changes

the Van der Hoeve-de Kleyn syndrome. Histologically the otosclerotic foci found in this disease are very active and the deafness as a

rule, rapidly progressive. There appears to be a congenital deficiency in the mesenchymal tissues of the body

Chondrodystrophia (Achondroplasia)—This is supposed to be caused by a checking or disturbance in growth of the cartilaginous tissues of the endochondral ossification.⁴ There is premature closure of the epiphyseal lines. Many sufferers are dwarfs who die early but some live to middle age. The changes in the labyrinthine capsule are confined to the endochondral layer. If mild there are only slight changes in the endochondral ossification and slight resulting anomalies in the cartilage remnants. If severe

tions of hormones, and by intoxications with salts of heavy metals. In the temporal bone, as a rule, only the periosteal capsule is affected whereas the endochondral and endosteal layers are not affected by these means. Wittmaack claimed to have produced otosclerotic foci in the otic capsule of chickens by producing a stasis in the veins of the adjacent areas. According to Altmann the changes described by Wittmaack represent a manifestation in the labyrinthine capsule of a generalized disease of the skeleton, which may be caused by faulty nutrition alone.^{5, 6} In spite of their histologic similarity to or even seeming identity with



Fig. 315.—Endochondral capsule in a newborn child having chondrodystrophia (achondroplasia). G O, Globuli ossi; S B, Skeinlike bone; W B, Weblike bone.

there are marked disturbances in the endochondral ossification and cartilage remnants.

An abnormal vascularization of the cartilage occurs with the formation of the so called 'cartilage marrow channels' followed by formations of areas of weblike fibril bone. The interior of the labyrinth is normal. The hearing is usually not affected and the condition, therefore, of no great importance to the otologist.

Experimental Alterations of the Labyrinthine Capsule—As of the skeleton as a whole, experimental alterations of the labyrinthine capsule may be produced by a great variety of means, such as deficiency or abundance of certain mineral salts or vitamins in the food, by injec-

tions of hormones, and by intoxications with salts of heavy metals. In the temporal bone, as far as is known these changes have nothing to do with true otosclerosis.

ENDOCRINOPATHIES AND METABOLIC DISEASES

It is known that many of the endocrine glands, particularly the pituitary, thyroid, and parathyroid glands, influence bone metabolism.

Certain pituitary tumors cause dwarfism from a general underdevelopment of the skeleton.

Congenital Athyrosis—This results from a congenital absence of the thyroid gland, which sets up the syndrome of congenital myxedema or myxedema and dwarfism, great speech and some hearing defects are frequent. The otic cap-

sule shows in its periosteal layer an osteosclerosis with slight underdevelopment of the endochondral and normal development of the endosteal layer of the labyrinth. It should also be noted that in acquired myxedema the hearing is usually unaffected.

Endemic Cretinism—This affection is endemic in alpine regions in varying degrees. It is characterized by dwarfism associated with skull deformities, mental and hearing disabilities (deaf-mutism), goiter, and certain skin abnormalities. The petrous bone is smaller than normal in size with a mastoid process which, although underdeveloped, stands out in exaggerated relief. The cavum tympani and its medial wall show a marked hypertrophy which narrows or may even occlude the labyrinthine window niches. The mucosa is thickened and contains remains of fatty or embryonic myxomatous tissue. The ossicles are deformed and frequently adhere to the adjacent bone.

Microscopically the petrous bone shows typical changes. There is an hyperostosis of the periosteal layer with weblike and lamellar bone, and indications that the process continues throughout life, which explains why it is found further developed with age. The endochondral layer of the capsule is quite normal but the endosteal layer shows focal defects, especially in the semicircular canals. The alterations in the membranous labyrinth are confined to Corti's organ and are slight, which may account for the absence of total deafness in these patients. Both nerve and obstructive deafness are encountered and the former is probably often, in part at least, central.

Diffuse Xanthomatosis—This disease is due to a dysfunction of the reticulo endothelial cells caused by disturbances in lipid metabolism. It occurs in two forms: primary generalized (essential), and secondary (symptomatic). The clinical manifestations of the first form depend predominantly on the type of the disturbance in the lipids. In Gaucher's disease kersin will be found in the tissues, in Niemann-Pick disease, phosphatides, and in Schuller-Christian disease, cholesterol. The secondary form may be associated with diabetes, liver affections, jaundice, or glomerular nephritis. Affections of the ear have occurred mostly in the first mentioned (idiopathic) forms. *Gaucher's*

disease does not have much effect upon the ear although the Gaucher's cells have been found in the marrow spaces of the petrous bone without any other change in the bone itself. *Niemann-Pick disease* (typical) has shown ear disorders in only a few cases. Typical Niemann-Pick cells were found in all parts of the ear including the marrow spaces of the periosteal layer, and in the perineural labyrinth. *Schuller-Christian syndrome* shows, in its typical form, defects of the skull, maplelike areas of decalcification, exophthalmos, diabetes, and hypercholesterinemia. In the soft tissues there is general presence of xanthoma cells. The ear is rather frequently affected, with suppuration, granulations, swelling of the mastoid and temporal bone, bony defects, and labyrinth symptoms. The roentgenogram clearly reveals the affected areas.¹

Ochronosis—This is a metabolic disorder showing dark pigmentation of the connective cartilaginous and bony tissues. The ear shows pigmentation in the tympanic membrane and in the endosteal and periosteal layers of the labyrinthine capsule and in the connective tissue of the labyrinth. The endochondral capsule does not show this pigmentation.

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FRANZ ALTMANN

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SUPPURATIVE LABYRINTHITIS

Suppurative labyrinthitis may be divided on the basis of origin into two main types: meningeal and tympanic.

Meningitic Labyrinthitis—This type results from extension of infection in the subarachnoid space. Invasion follows two routes: (1) Along the nerve bundles into the modiolus of the cochlea chiefly, thence directly into the perilymphatic space.¹ Nerve bundles and spiral ganglia are primarily involved (neurolyabrin-

mastoid, or petrous pyramid). It may be divided into two forms: circumscribed and diffuse.

Circumscribed Labyrinthitis (Perilabyrinthitis)—This is an inflammation localized mainly to the perilymphatic space beneath an area of osteitis and resorption of the bony capsule. It is usually due to cholesteatoma, more rarely to granuloma or tumor. Usually the outer limb of the horizontal canal and occasionally a vertical canal is involved. Localized resorption of the capsule of the vestibule and cochlea has been found on histologic examination.

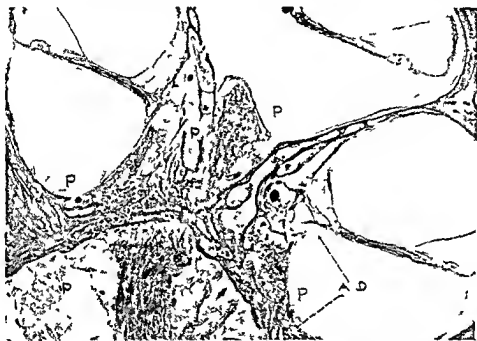


Fig. 316—Microphotograph showing the early stage of meningeal invasion of the cochlea from the internal meatus in a diabetic patient. Pus cells (P) pass from the subarachnoid space along nerve bundles into loose areolar tissue of the modiolus, thence into the perilymphatic space upon which the tissue borders directly at some points (A, D). (From Perlman H. B. and Lindsay J. R. Relation of the Internal Ear Spaces to the Meninges, Arch. Otolaryng. 29.)

thitis) (Figs. 316, 318). (2) Along the cochlear aqueduct into the scala tympani (Fig. 317).

In the suppurative stage of meningeal labyrinthitis the membranous labyrinth and neural elements undergo destruction (Fig. 319).

Meningitic labyrinthitis is a frequent necropsy finding. The healed form with permanent and usually total loss of function has been common after meningococcus and recently has been seen in cured pneumococcus meningitis.

No therapy is indicated. Since loss of function is usually bilateral the problem is one of education and rehabilitation.

Tympanic Labyrinthitis—This type results from invasion by suppurative in the middle ear,

Circumscribed labyrinthitis is common in congenital syphilis with osteitis or osteomyelitis of the labyrinthine capsule.²

The chief symptom is recurring vertigo which may often be induced by sudden movements and sometimes by pressure over the tragus. Spontaneous nystagmus, directed to either side (usually to the affected ear), is present during attacks. The fistula reaction may usually be elicited by means of a Siegle's speculum or a rubber bag and olive tip fitted into the external canal or by pressure with the finger over the tragus. Vertigo and nystagmus on increased or decreased pressure constitute a positive reaction. Reversal of pressure reverses the nys-



Fig 317 —Microphotograph showing the early stage of meningitic invasion of the cochlea through the cochlear aqueduct. Pus cells (P) are collected at the mouth of the cochlear aqueduct. The upper coils of the cochlea contained no pus cells (From Perlman H B and Lindsay J R. Relation of the Internal Ear Spaces to the Meninges. Arch. Otolaryng 29)

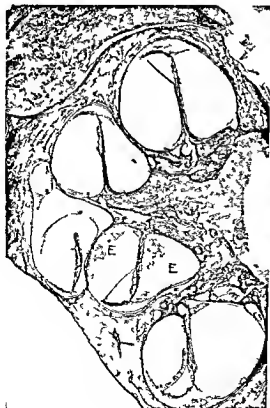


Fig 318



Fig 319

Fig 318 —Microphotograph showing the early serofibrinous stage of meningitic labyrinthitis. Serofibrinous exudate (E) has collected mainly in the middle coil. Pus cells have invaded the modiolus but not the labyrinthine fluids.

Fig 319 —Microphotograph showing the diffuse purulent stage of meningitic labyrinthitis. Pus cells are diffusely distributed throughout the cochlear spaces and the modiolus. Destruction of the cochlear duct has already occurred in the basal coil.

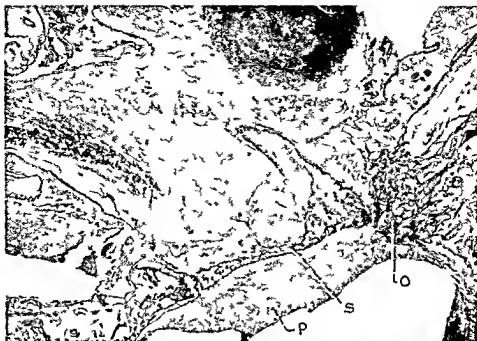


Fig 320—Microphotograph showing the invasion of the labyrinth through the oval window in the early stage of an acute otitis media in a diabetic. Pus (P) fills the vestibule. The annular ligament has been destroyed between the stapes footplate (S) and the labyrinth capsule in which there is a small otosclerotic focus (O). The middle ear is filled with granulation tissue and pus.

tagmus. Negative reaction to the test may be obtained when there is excessive granulation or fibrous tissue formation. Other tests for vestibular function are not essential. Cochlear function is present and impairment varies in degree.

The clinical diagnosis is based upon one or more attacks of labyrinthine vertigo and nystagmus with a positive fistula reaction and the presence of cochlear function.

As a complication, diffuse suppurative labyrinthitis may occur (1) during an acute exacerbation of the suppuration (2) following surgery, or (3) by slow progression.

The treatment usually indicated is a mastoid operation, either complete or, if the membrana propria is intact, a modified radical. The cholesteatoma matrix should not be removed from the medial wall of the antrum and aditus. The site of the fistula should not be curetted and firm packing should be avoided. To protect against diffuse labyrinthitis, sulfonamide or penicillin therapy (sufficient dosage to insure adequate blood concentration should be given) before and after operation is advised.

The prognosis is good if careful surgery has been performed. The function is usually preserved or improved. The bony fistula may close or remain permanently patent.

Diffuse Labyrinthitis—Diffuse labyrinthitis



Fig 321—Microphotograph showing the erosion of a semicircular canal in meningitic labyrinthitis. Osteitic resorption of the capsule may occur from without inwards in middle ear and petrosal suppuration or from within outwards in meningitic labyrinthitis. Suppuration in the superior canal (S.C.) has caused resorption of the capsule (R) and osteitis spreading along vascular channels to the pneumatic spaces.

may occur (1) in a healthy labyrinth or (2) following circumscribed labyrinthitis. The organisms are usually hemolytic streptococci, pneu-

In acute middle ear suppuration, invasion may occur by way of the windows (Fig. 320), or by erosion of the bony capsule. The former



Fig. 322—Microphotograph showing invasion of the meninges of the internal meatus from labyrinthitis. Soft tissue parts of the cochlea are destroyed. A collection of pus (P) has formed in the base of the modiolus. Note thickened dura mater, and granulation tissue (D).

mococci, staphylococci, or, more rarely, the tubercle bacilli. In chronic middle ear suppuration, diffuse invasion occurs usually during an acute exacerbation, but sometimes by chronic

occurs in the serous or early suppurative stage or following injury to the stapes. The latter process occurs in the later stages of acute suppuration. Petrositis is a frequent predisposing

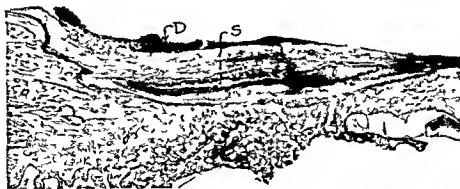


Fig. 323—Microphotograph showing an abscess formation in the saccus endolymphaticus in a case of labyrinthitis in a diabetic. Pus is seen in the saccus (S) and on the inner surface of the dura (D). The adjacent bone shows a diffuse osteitis.

or periodic extension. The predisposing factors are (1) pneumatization of the petrous pyramid, (2) cholesteatoma, and (3) systemic disease, such as uncontrolled diabetes mellitus.

factor. Erosion of the bony capsule occurs directly or by progression along bony vascular channels to reach the endosteum (Fig. 321). In simple chronic suppuration, invasion occurs

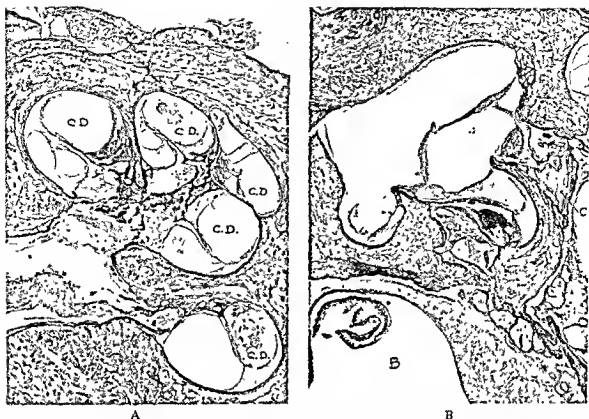


Fig. 324.—Chronic diffuse labyrinthitis in case of middle ear cholesteatoma with acute exacerbation and jugular bulb thrombosis. A, Microphotograph of the cochlear section showing ectasia of the cochlear duct (C.D.) with recent fibrinous exudate. There is new bone formation in the scale vestibuli and modiolus, irregular fibrosis in the perilymphatic spaces and modiolus, and extensive but incomplete degeneration of neural elements. B, Microphotograph showing the oval window niche filled with cholesteatomatous debris (C) and the round window niche with inflammatory products. The jugular bulb (B) is filled with pus and granulation tissue. The scala tympani is partly filled with new bone. The endolymphatic spaces have been intact although distorted until the terminal exacerbation.



Fig. 325.—Microphotograph showing obliteration of the inner ear spaces by new bone and fibrous tissue following labyrinthitis in a rabbit. The cochlea (C) has been completely obliterated. Remnants (R) of the membranous labyrinth are seen in the vestibule surrounded by fibrous tissue and new bone. Stapes (S).

frequently from suppuration in the pyramid while cholesteatoma usually leads to invasion from the mastoid and tympanum

Infection may invade the cranial cavity by way of the internal meatus (Fig 322), through extension from a saccus endolymphaticus abscess (Fig 323), or, possibly, along the cochlear aqueduct

Healing occurs by fibrosis and new bone formation Ectasia of the cochlear duct and degeneration of neural elements may occur (Fig 324, A and B) or destruction of soft parts followed by obliteration by new bone (Fig 325)

Chronicity and sequestration is predisposed by undrained petrosal suppuration

Diffuse labyrinthitis may be divided into three clinical groups (1) serous, (2) acute suppurative or destructive, and (3) chronic diffuse or destructive

DIFFUSE SEROUS LABYRINTHITIS—This group includes clinically the early stages of invasion with vestibular and cochlear symptoms, but demonstrable function Transient forms occur in the serous stage of otitis media or following mastoidectomy without loss of cochlear function Depression of cochlear function indicates an impending purulent stage The serous stage may be brief or last several days

The histologic picture is probably that of serous or mild serofibrinous exudation with beginning cellular infiltration (See Figs 316 and 318)

Symptoms may increase rapidly or slowly Rotational vertigo, nystagmus usually to the diseased side, nausea, perhaps vomiting, and beginning nerve deafness indicate the onset Vertigo may be postural in early stages Nystagmus may change to the sound side Fever and pain may occur but are due to the surrounding suppuration Bone conduction tests with masking are useful to indicate cochlear damage in early stages The simplest qualitative test is the loud voice with the Barany noise apparatus in the good ear With demonstrable hearing the vestibular tests are not essential

In the *diagnosis*, nonsuppurative labyrinthine disease or neuritis of the eighth nerve may be confused with diffuse serous labyrinthitis Clinical or roentgen ray evidence of suppurative middle ear disease is necessary for diagnosis

As a complication, invasion of meninges may occur, usually during the purulent stage

Medical treatment consists in absolute rest, the administration of a mild barbiturate, and

chemotherapy Sulfadiazine at present offers the greatest protection to the meningeal and labyrinthine spaces Intravenous medication assures control of fluid intake and dosage

In the *surgical treatment*, when the onset occurs during the serous or the first days of the suppurative stage of acute otitis media only middle ear drainage is required Onset after a week or more of acute suppuration or during chronic suppuration is an indication for roentgen ray examination of the mastoid and pyramids A mastoidectomy with drainage of any petrosal suppuration is usually indicated In such cases, surgery should be prompt in order to save cochlear function The labyrinth operation is not indicated

As to *prognosis*, return of useful function occurs if the deafness has not been profound Partial function may return after apparent complete loss indicating that the disease had therefore not reached the purulent stage

ACUTE DIFFUSE SUPPURATIVE OR DESTRUCTIVE LABYRINTHITIS—Acute diffuse suppurative labyrinthitis with complete loss of vestibular and cochlear function may develop abruptly or follow a serous stage The inner ear becomes diffusely infiltrated with pus cells with destruction of soft tissue structures

The *symptoms*, vertigo, nausea, and vomiting, are severe or moderate depending upon the rapidity of the onset Nystagmus is quick to the sound side Cochlear function is abolished Absence of caloric responses and failure to hear a loud shout at the ear with the good ear masked by a Barany noise apparatus are the simplest tests for the absence of function Rotation tests are impractical in acute stages but after vertigo has passed the after nystagmus following rotation to the sound side is reduced to about five as compared to a normal of about twenty seconds Fever and pain may occur but are suggestive of an undrained focus in the bone or intracranial extension Roentgen ray examination of the mastoid and pyramids is indicated Cerebrospinal fluid examination is indicated in the presence of signs of meningeal irritation

In the *differential diagnosis*, acute destruction of labyrinthine function due to nonsuppurative disease is excluded by local and roentgenologic evidence of suppuration

In pre sulfonamide years, diffuse meningitis (resulting in fatality) occurred as a *complication* of this type of labyrinthitis in a high percentage of cases^{3, 4} and, occasionally, cerebellar abscess

The primary objective in the *treatment* is the prevention of an intracranial extension. In presulfonamide years the only resource for prevention of meningitis was drainage of the labyrinth. Chemotherapy now provides more adequate protection and the operation has become of secondary value. Experience indicates that with adequate chemotherapy the appearance of meningeal signs during treatment will be rare.

In the presence of frank meningitis recovery depends at the present time upon the response to sulfonamides or penicillin. Drainage of the relatively small collection of pus in the labyrinth is probably of minor importance.

Medical treatment consists in absolute rest, mild hypnotics, and chemotherapy.

Mastoidectomy and drainage of any petrositis is usually indicated when the onset of labyrinthitis occurs in late stages of acute or during chronic suppuration. Chemotherapy obviates the risk of spreading infection during an acute exacerbation.

The labyrinth operation is indicated (1) when early meningeal signs have persisted, or have made their appearance during or following chemotherapy, (2) following a recognized dislocation of the stapes in an active labyrinth during mastoidectomy, or (3) in the presence of a purulent labyrinthine fistula, to promote healing. Tuberculous otitis media and labyrinthitis rarely produce an intracranial extension,⁵ and the labyrinth operation is, therefore, not an essential.

CHRONIC DIFFUSE OR DESTRUCTIVE LABYRINTHITIS—This follows the acute suppurative stage. The vestibular symptoms have passed and function is absent. Cessation of discharge and healing may occur or purulent discharge may persist indicating suppuration in the surrounding bone, or in the labyrinth or both. Sequestration of small or large parts of the labyrinthine capsule may occur. Suppuration in the petrosa is a frequent cause.

Deafness and vertigo are the chief *symptoms*. Deafness is permanent. Slight vertigo on quick movement and mild spontaneous nystagmus to the sound side, with fixation eliminated, persists for several months. Caloric response is absent. The differential in postrotatory nystagmus is reduced. Facial paralysis is a common sign.

Treatment is mainly surgical. With persistent suppuration, roentgen ray examination of the mastoids and pyramids is indicated and a radical mastoidectomy is performed. Any petrosal

suppuration should be drained, removing the superior canal if necessary for this purpose. Loose sequestra may be removed if not involving the facial canal. Chemotherapy is indicated pre- and postoperatively for its protective effect. In the presence of a facial paralysis the facial canal should be uncapped in the affected region.

Healing is slow in the presence of sequestration or chronic osteomyelitis.

THE LABYRINTH OPERATION

The primary objective of the labyrinth operation is to provide drainage from all areas. To accomplish this two procedures are essential: (1) Removal of the stapes and the promontory between the oval and round windows. This provides drainage from the cochlea by way of the basal coil and from the semicircular canals by way of the vestibule. (2) Exposure of the posterior fossa medial to the sigmoid sinus and elevation of the dura mater over the niche for the saccus endolymphaticus. Invasion of the meninges from an abscess in the saccus may thus be prevented. Additional drainage may be provided by an opening into the prominence of the posterior vertical and horizontal canals.

In the presence of a perilyabyrinthine petrositis removal of the posterior part of the superior canal may aid the surgical approach.

Further interference with the cochlea is not justified since injury to the modiolus facilitates extension of infection to the subarachnoid space.

The desired objectives may be attained by the Hinsberg operation with modifications.

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TUBERCULOSIS OF THE MIDDLE EAR

Tuberculosis of the middle ear is nearly always secondary to other tuberculous lesions and particularly to pulmonary tuberculosis. It is often masked by secondary infection with staphylococci, or even streptococci, from the external auditory canal as soon as there is a perforation in the membrana tympani. The typical appearance of a tuberculous disease is thus obscured. This probably explains the infrequency with which the diagnosis is made.

In children the middle ear disease may be secondary to tuberculosis of the lungs, lymph nodes, bones, or joints.

The history of the disease has been reviewed by Briggs,¹ Spencer,^{2, 3} and others.

Etiology.—The disease is usually caused by human tubercle bacilli. Bovine tubercle bacilli may be the cause in children or adults who drink unpasteurized milk. When J. S. Fraser of Edinburgh, Scotland, was in the United States in 1923 he showed many slides of tuberculosis of the temporal bones of children who had died from tuberculosis. When asked why he had so many specimens of tuberculosis he said, "The milk in Scotland is not pasteurized. We have entirely too much bovine tuberculosis in children."

The bacilli may gain entrance to the middle ear through the eustachian tube or through the lymph or blood stream. Much has been said in recent years about the latter avenues of infection. In children with tuberculosis of bones or joints the lymph and blood streams must be the avenues. In adults with pulmonary tuberculosis, infection certainly enters through the tube in some patients. Briggs¹ believed the recumbent position of patients of all ages with pulmonary tuberculosis favored infection through the tube.

Pathology.—Microscopically, epithelioid and giant cells are found. The latter show caseation necrosis. The disease belongs to the group of granulomas. The finding of tubercle bacilli by staining is the final proof of the cause of the disease.

The gross lesions show pale granulations with relatively little pus, which is thin.

Grimmer,⁴ according to Briggs,¹ found absorption or softening of the intima at isolated points through which the invasion extended to the dermal layer, thus accounting for the

existence of multiple perforations" (See Fig 326, A).

Symptoms.—The disease is nearly always chronic from the beginning and usually there are no symptoms. The ear may or may not feel stuffy or full. A small amount of thin pus may be accidentally discovered by the patient in the external auditory canal as the first and only symptom. Before spontaneous perforation takes place the membrana tympani is pale and two or three milium tubercles may be faintly seen in the drum head. When these rupture there are multiple perforations. The disease is remarkably free from pain. Briggs¹ said, "as long as the condition remains characteristically tuberculous and the ravages of pyogenic microorganisms are absent the process is painless" (See Fig 326, B).

Staphylococci, from the skin of the external auditory canal, soon invade the perforations to enter the middle ear. More pus is formed as soon as the staphylococci begin to grow. After months of secondary infection the multiple perforations coalesce to form one larger perforation. This explains why so few otologists see more than a single perforation. The perforations are usually central, but may be peripheral (See Fig 326, C).

Patients with pulmonary tuberculosis are rather susceptible to upper respiratory infections. A streptococcal pharyngitis, tonsillitis, or bronchitis may extend to the tuberculous middle ear just before or soon after spontaneous rupture of the membrana tympani has occurred from tuberculosis. The multiple perforations are converted into one large perforation in a very few days instead of in months. The pneumococcus may produce the same picture (See Fig 326, D).

Diagnosis.—The pallor of the drum head is typical of the secondary anemia of the tuberculous. The pus is thin and usually scant, the perforations multiple or, later, single. The granulations are pale. The presence of pulmonary tuberculosis or tuberculosis of other organs, whether the disease is active or inactive, should suggest the possibility or even the probability of a tuberculous otitis media. A culture often shows only staphylococci. Pus drawn from the middle ear through the perforation, by suction, may reveal tubercle bacilli. A guinea-pig inoculation may be positive for tuberculosis. A biopsy of the granulation tissue may establish the diagnosis. It is very difficult to ob-

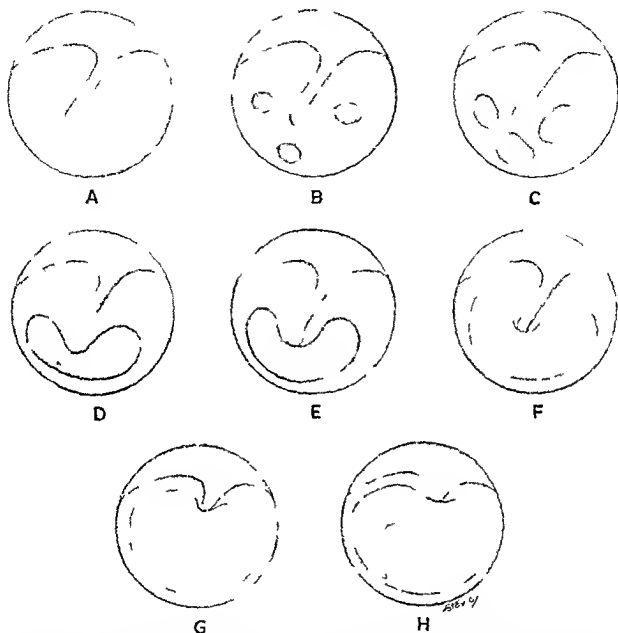


Fig. 326—A There are three tiny tubercles of the membrana tympani. These do not show very distinctly in the living patient and can easily be overlooked. B Multiple perforations are present where the tubercles were formerly. These represent spontaneous ruptures of the membrana tympani without pain at any time during the course of the tuberculous otitis media. C, The staphylococcus invades the middle ear through the perforations, from the skin of the external auditory canal. The secondary invaders produce more pus and gradually increase the size of the perforations. D As a result of the secondary infection the three perforations have coalesced to form one large perforation. This change takes place gradually over a period of months. E The longer the disease lasts the larger the perforation becomes. At this stage the primary infection has been masked by the secondary infection with the staphylococcus. The multiple perforations now form one. F After months of discharge of pus, pale granulations form over the inner wall of the tympanum. Acute exacerbations from the streptococcus, pneumococcus, or the staphylococcus make these red. G Necrosis of the manubrium with gradual enlargement of the perforation and more granulation tissue mean more and more impairment of hearing. H The final stage of the tuberculous otitis media is reached after months or years. Almost the entire membrana tympani has been destroyed. There is an abundance of pale granulations and the hearing is very poor.

tain enough tissue for a biopsy specimen unless there is a polyp or there are abundant granulations (See Fig. 326 E.)

For cultures Haskin⁵ recommended the use of Petroff's medium, which was the original medium used by Dr. James G. Dwyer at the

Manhattan Eye and Ear Hospital, and advised that it be used with Miller's later medium, made from the spleen and lymphatic glands of animals. If tubercle bacilli are present a growth is easily obtained in from ten to twenty days. Haskin advised the use of antiformin before staining the smears for examination (See Fig 326, F)

Prognosis—Tuberculous otitis-media is not easily cured. The disease is usually chronic and mild. It resists treatment of any and all kinds. The ear may become dry only to discharge again with each upper respiratory tract infection. Complications are rare in the chronic cases and danger to life is almost nil. However, there are acute cases of tuberculosis of the middle ear, complicated by meningitis, which terminate fatally. Acute tuberculous otitis media may invade the internal ear and the resulting tuberculous labyrinthitis is soon followed by a tuberculous meningitis and death. Also, there are rare acute toxic fulminating cases which are a threat to life (See Fig 326, G)

Treatment—Myringotomy should be performed if the otologist sees the patient before spontaneous rupture has occurred. The painless, symptom free course of the disease usually prevents the otologist from examining the patient's ear prior to spontaneous rupture.

The ear should be kept clean. One or 2 per cent iodine in boric acid insufflated in the ear every few days is helpful. Boric acid (20 grains to 1 ounce of ethyl alcohol) usually reduces the discharge. Silver nitrate solution, on a cotton wound-applicator, may be applied to the granulations, at first in strengths of 5 per cent, gradually increasing to 10 or 20 per cent. If polyps form, they should be removed with a snare. They are rare, however, in tuberculosis of the middle ear. Mercurochrome (2 per cent aqueous solution) may be dropped in the ear. Mercurial solutions may be used to lessen the odor if it is foul. Gentian violet may help some patients. The treatment must be used which, in the judgment of the otologist, is best suited to each patient. It should be changed from time to time as the middle ear disease changes (See Fig 326, H)

Ultraviolet light has a useful place in the treatment of tuberculous otitis media. Direct sunlight is best. Chapman⁶ has devised a mirror made of magnesium and aluminum which reflects a very high percentage of the ultraviolet rays from the sun into the the middle ear. An

ordinary mirror is not satisfactory because the glass absorbs too many of the ultraviolet rays and reflects chiefly the heat rays.

Tuberculin has an important place in the treatment of tuberculosis. It is not a panacea, however. Patients with acute tuberculosis or with high fever due to the chronic disease are rarely good subjects for tuberculin therapy, nor are highly excitable, emotional, and unstable patients. Tuberculin is not a useful or reliable remedy in the hands of a tyro. Much of the disrepute voiced against it comes from physicians and patients who expected immediate results. Tuberculin must be used for months or not at all. Each physician, experienced in the administration of tuberculin, has certain forms he is familiar with and he can use these best. Changing frequently from one form of tuberculin to another is disappointing and can be even dangerous. Old tuberculin, new tuberculin, and bacillary emulsion are a few of the reliable forms.

Local, focal, and general reactions should be avoided. These worry the patient, the relatives, and friends. They tend to defeat the benefits to be obtained by using tuberculin. Very mild local, focal, or general reactions may be used to determine the best dosage for each patient. Any beginner can produce a severe general reaction and he may keep the patient from ultimately recovering.

Most patients with tuberculosis of the middle ear have other organs involved, such as the lungs. Tuberculin cannot be given, therefore, just for the ear. The whole patient must be treated and not just the ear. In cases of pulmonary tuberculosis, teamwork by the chest expert and the otologist will produce the best results.

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SYPHILIS OF THE EAR

Syphilis of the External Ear—The external ear, as well as other parts of the body, may be the site of a syphilitic lesion. The auricle or the canal may be involved. A primary lesion is extremely rare, but a few have been reported. Secondary lesions of all types, including condylomas, are more frequent, and in some cases, if untreated, may result in great deformity of the auricle. In tertiary syphilis there may be gummatous involvement of the canal wall, with occasional necrosis and sequestration of the bone, in rare cases there may be ulceration of the canal wall. Cholesteatoma may arise from proliferation of the squamous epithelium during the healing process. In all types of syphilitic lesions of the external ear, timely and adequate treatment produces complete healing.

Syphilis of the Middle Ear.—Syphilis of the middle ear is of two kinds. It may be a sequela to a secondary syphilitic lesion or a tertiary lesion (gumma), located in the nasopharynx or at the orifice of a eustachian tube. Inflammation surrounds the lesion, and ascends into the middle ear, producing an otitis media, either catarrhal or suppurative. Even mastoiditis has been reported. Such entry of the syphilitic infection into the throat or eustachian tube has been attributed to the use of infected instruments in eustachian tube catheterization or nasopharyngeal treatment. Middle-ear involvement of this kind is rare.

More common in middle ear involvement, as a part of the involvement of the internal ear, is otolabyrinthitis. Rarely, these pathologic processes may be present in the middle ear without involvement of the internal ear. Sometimes in the second stage of syphilis, and occasionally in the third stage, there is presented a picture of a painless catarrhal, or even a mild suppurative, otitis media. Such a picture, together with other findings, strongly suggests syphilitic involvement. More often, however, no visible signs of the infection are present.

The pathologic lesions of the middle ear include osteomyelitis, perostitis, and round-cell infiltration. Aural connective tissue may become hyperplastic and the mucosa may be thickened. New bone is often deposited. Necrosis and sequestra may be found. Sometimes there is ankylosis of the ossicles to each other and to the adjoining bone. However, we do not find the stapes ankylosed in the oval window.

All recent workers agree that syphilis is not a cause of otosclerosis.

Syphilis of The Internal Ear—Syphilis of the internal ear has been divided into four classes: (1) hereditary, early, (2) hereditary, late, (3) acquired secondary syphilis, and (4) acquired tertiary syphilis.

Symptoms—The classical picture in syphilis of the internal ear is one of high tone deafness of sudden onset and rapid progress, increasing day by day. Within a period of a few days to a few weeks there is a marked or complete deafness. Usually there are vestibular symptoms, especially tinnitus, as well. However, there may be great variations in this picture, e.g., gradual progress of the deafness, lasting over months or years, no vestibular symptoms, or vestibular symptoms without deafness. The type of deafness may vary, too. There is usually no pain, and few, if any, signs of middle ear involvement. The deafness is usually permanent, while the tinnitus and vertigo tend to disappear after the complete destruction of the labyrinth.

The hereditary syphilis begins in one ear, but ultimately affects the other ear as well, while the acquired disease, although more often bilateral, may be unilateral. The early hereditary is present in infancy, with deaf mutism resulting.

Moore states that the hereditary syphilitic deafness is an isolated phenomenon and not connected with other nervous disorders. In the acquired form there may be coincident paralysis, and this form is not infrequently associated with *tuberculous*.

Involvement of the eighth nerve is sometimes the first indication of neurosyphilis. Ruskin and Hyslop state that syphilitic labyrinthitis can precede all other general manifestations of syphilis and can appear within seven days of the onset of the primary lesion. On the other hand, the symptoms may not occur until years after the beginning of the tertiary stage. Usually, however, they are manifest at the end of the secondary or the beginning of the tertiary stage. In congenital syphilis one sees involvement at birth and in early childhood (the so-called "early cases") and also between the ages of eight and fifteen (the so-called "late cases"). Some of this latter group have been reported as early as four years, and Rodger reported two patients, one of forty-five and one of forty-seven years.

Incidence—The incidence of syphilitic deaf-

ness has been the subject of much controversy. It used to be thought that many cases of high tone deafness were due to syphilis. Rodger found 500 cases in ten years of his practice. Alexander and Krassing, quoted by Ciocco, say that 20 per cent of patients with latent syphilis, and 69 per cent of those with central nervous system syphilis, acquire some form of nerve deafness. Ciocco states that deafness is twice as frequent in neurosyphilis as in other forms.

Recently there has been contradicting evidence presented.

Ciocco says, "Syphilis does not cause 'nerve' deafness more often than any other general systemic disease."*

Bunch states, "The loss of acuity for tones of high pitch with advancing age is a constant factor in patients in whom a clinical diagnosis of arteriosclerosis, etc., and syphilis has been made."

Stokes states that deafness is present in 2 per cent of patients with early secondary syphilis, in 10 per cent of those with hereditary syphilis, and from 1 per cent to 4 per cent in patients with tabes dorsalis. He believes that, in eighth nerve deafness due to hereditary syphilis, there are probably more females than males affected, although Keidel and Kemp, mentioned by Stokes,¹ find the opposite to be true. There is probably an equal division in the acquired form.

Jones feels that the nervous system is attacked in greater than 5 per cent of syphilitics in general, while some reports run from 7 per cent to 48 per cent. He also feels that the eighth nerve is more likely to be affected than any other part of the nervous system.

Goodhill reports that "syphilis is not an important cause of deafness."

We can conclude from these facts that, in the average syphilitic, specific deafness is rare, and that if deafness is present it is probably due to other causes. On the other hand, in neuro- and congenital syphilis the condition occurs fairly frequently.

Pathology.—Syphilis of the internal ear includes involvement of parts of the brain connected with the eighth nerve, the eighth nerve itself, its ganglion, the labyrinth, and the organ of Corti, together with the vessels and the bony and fleshy structures thereabouts. Most any

one of these structures, or a combination of them, may be involved. Likewise, a lesion in one part of the ear may heal, while another becomes active. The result is a varying of both pathologic and clinical pictures.

Rodger speaks of syphilitic basal meningitis, meningoneuritis of the eighth nerve, and otolabyrinthitis.

Crowe *et al.* found no difference in the cochlear lesions that were possibly due to syphilis from those that were not.

Mayer and Fraser studied cases of late congenital syphilis. They found, in general, that there was in the cochlea and vestibule a serous labyrinthitis, which resulted from a gummatous osteitis and periostitis. Atrophy of the organ of Corti and other parts resulted. In the semicircular canals they found a productive periostitis, which led to a gradual decrease in the size of the lumen, the endolymph space, however, usually remained open. The nerve tissue was found to be involved, and there was seen both bone destruction and new growth.

In a study of eight early hereditary and eight late acquired cases, Goodhill finds the following distinct lesions occurring constantly:

- 1 Productive periostitis (syphilitic) producing in vasive fibrosis followed by ossification of the perilymphatic and endolymphatic channels of the vestibular apparatus. Malformations of the bony canals are also produced.
- 2 Bony abnormalities of the stapes especially of the foot plate.
- 3 Atrophy of the neuroepithelial elements of the cochlear and vestibular systems.
- 4 Lymphocytic (small round cell) infiltration of the cochlea and spiral ganglion (miliary gummata).
- 5 Vascular changes consisting of diapedesis in prenatal cases and obliterative endarteritis in acquired cases.

Diagnosis.—In syphilis of the internal ear the Wassermann blood test is generally unreliable, the reaction being positive in a large percentage at an early age, and becoming less positive as the age increases. In the spinal fluid Wassermann the results are almost always negative in hereditary syphilis. In the acquired form they are usually positive, but like the blood Wassermann, this test is less reliable as the age of the patient increases.

Late hereditary syphilitic deafness is usually accompanied by other stigmas of the disease. Interstitial keratitis is the most frequent of these. Others include Hutchinson's teeth, scars in the pharynx, and septal perforations. The

* This study was made upon patients with syphilis and deafness, but there was no evidence that the syphilis was the cause of the deafness.

well known Hutchinson's triad consists of labyrinthitis, notched teeth, and interstitial keratitis. These often make the diagnosis of syphilis positive when the Wassermann reaction is negative.

THE TYPE OF DEAFNESS—Syphilitic deafness has been said to take the following forms: (1) loss of acuity of high tones and lowered upper limit, (2) loss of acuity of all tones by air conduction and impaired bone conduction, (3) shortened bone conduction with normal air conduction.

Crowe *et al* state, "Syphilis does not cause high tone impairment without, at the same time, involving low tone perception."

Ciocco feels that "high tone loss is no more frequent in syphilitics than in non-syphilitic patients."

Goodhill concludes, and agrees with Ciocco and Bunch, "There is no specific type of hearing loss associated with syphilis, in middle aged patients, especially."

Bunch reports a case of neurolabyrinthitis due to syphilis where there is a gap in the audiogram at 1500 double vibrations. This gap agreed with his histologic studies on the case.

Drury analyzed types of audiometric curves with reference to syphilis and notes the following: "There are a surprising number of syphilitics showing the types of curves where there is a loss of the high tones. A few cases of strongly suspected syphilitic deafness, however, showed other types of audiometric curves." He also noted that syphilitics tended to have the greatest loss at 4096 double vibrations. He believes this to be the earliest audiometric manifestation of syphilitic deafness and that such a dip indicates permanent involvement.

Ciocco found, in a large group of hospital patients, that the 4096 dip was no more frequent in syphilitics than in nonsyphilitics.

RINNE'S TEST AND BONE CONDUCTION TEST—Again the later work indicates that there is no greater loss of bone conduction as compared to air conduction than would be present in any kind of nerve deafness.

Drury feels that the presence of a negative reaction in Rinne's test in some cases of unilateral involvement is due to the fact that, as one ear becomes more deafened, the sound is referred (by bone conduction) to the opposite ear, and hence, in the poor ear, bone conduction is greater than air conduction.

Mayer and Fraser explain an occasional negative reaction to Rinne's test by showing that, histologically, the lesion may be that of ankylosis of the ossicles (except the foot plate of the stapes). Such a lesion would account for the negative reaction.

VESTIBULAR REACTIONS—Jones states that in syphilitic involvement of the labyrinth there is a rapid reduction in the vestibular function, as evidenced in the turning tests.

Mayer and Fraser and many others find that labyrinth tests show striking discrepancies. They explain these variations anatomically, pointing out that the lesion may be localized to a variety of different places in the internal ear and labyrinth, while other parts may be entirely untouched. The vestibular tests may change from time to time, again because the lesion may change its location.

However, most agree that as the deafness becomes marked the functional labyrinth tests show a likewise marked diminution of activity.

It has been shown that in some cases there is a so called vestibular paradox—loss of reaction to rotation and retention of the caloric reaction (and sometimes vice versa). Mayer and Fraser explain this in this manner: The caloric reaction depends upon circulation through the entire horizontal semicircular canal, while the much stronger rotation stimulus may cause displacement of fluid also in the other semicircular canals and the saccules. The cases showed thickening of the endolymph in the horizontal canal, which explains why displacements could be caused by rotation but not by a difference in temperature. They state that "positive caloric reaction with negative rotation test would be inexplicable histologically."

The same authors explain anatomically the fistula symptom, first described by Hennebert, wherein there is no perforation of the tympanic membrane and no suppuration of the middle ear. They depict cases in which there is a labyrinthine gap, due to osteomyelitis, gumma (leading into the marrow space), or a gummatous infiltration of the stapedial ligament, allowing for its abnormal mobility.

Differential Diagnosis—Some cases of syphilitic disease give a picture closely resembling tubercular otitis—slight discharge that is painless. If the tubercle bacillus or the spirochete can be found, the diagnosis is made. Biopsy is often necessary. Often there is great difficulty in distinguishing between a miliary gumma and a

miliary tubercle. Special staining methods were used for this by Mayer and Fraser.

The rapid and continuous reduction of vestibular and cochlear function in syphilis aids in differentiating it from mumps and other diseases, in which, although there is a reduction, the reduction remains the same.

Ruskin and Hyslop point out the similarity between concussion and syphilitic deafness. Either may be manifest by headache, vestibular symptoms, and deafness. One must depend upon other characteristic signs and symptoms of syphilitic involvement to distinguish between the two.

In arteriosclerosis and chronic progressive deafness there is a slow progressive deafness compatible with age.

Occupational deafness often affects but a few tones in the scale. It has a gradual onset, and there is a history of exposure to certain loud tones or noises. Sometimes but one ear is involved, as in Hunter's deafness.

Foci of infection produce a gradual deafness. The focus is usually found.

In otosclerosis there is much tinnitus, but the history, the gradual onset, and the presence of a negative reaction to Rinne's test distinguish it.

Prognosis and Results of Treatment—The prognosis in syphilitic deafness, without treatment, is always poor. With treatment there is some hope for the patient. Authors disagree, however.

With only the labyrinth involved, Drury feels that the prognosis is better than when the cochlea, too, is involved.

Ciocco found that treatment made no appreciable difference in his series of cases. He quotes Alexander and Benario, who found that the hearing in from 50 per cent to 83 per cent of the patients was improved or completely restored by proper treatment.

Rodger and Smith (quoted by Moore) find no improvement from treatment in congenital syphilitic deafness. Navarro, in discussing Rodger's work, disagrees.

Moore finds that, with the proper treatment of early meningeal neurosyphilis, the hearing in as many as 50 per cent is improved, but in the later cases, and also in the congenital, the best to be hoped for is an arrest of the process.

Herxheimer's reaction, manifest by sudden deafness, following specific treatment has been reported as occurring. However, most feel that the treatment is not responsible when a sudden

decrease in hearing occurs and that its advent should call for the institution of more intensive treatment.

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FRACTURE OF THE TEMPORAL BONE

A fracture of the temporal bone is an interruption or a break of the continuity in any part or in various parts of the bone. It may be either partial or complete, unilateral or bilateral. Fractures may also be classified in the order of their direction and may be longitudinal, oblique, or transverse (See Fig. 327). Fractures of the temporal bone may involve either the squamous, the mastoid, or petrous portions.

They may be limited to the temporal bone or be accompanied by fractures of the skull involving the vault or the posterior fossa or both

factors may be occupation modern industry, and warfare



Fig 327—Anatomical photograph showing longitudinal and transverse fracture of the petrous portion of the temporal bone

Etiology—Fractures resulting in intracranial damage are almost always traumatic in origin and result from direct blows contrecoups or



Fig 328—Roentgen ray photograph showing transverse and longitudinal fracture of the squamous and petrous portions of the temporal bone

sudden jarring The mechanical factors of a head injury may be the movement of the skull and brain and the type of impact The predisposing



Fig 329—Roentgen ray photograph showing transverse fracture through the petrosa with involvement of the squamous portion of the temporal bone

Anatomy—The temporal bone is strategically situated in the skull occupying a space between the middle and posterior fossae As its intricate



Fig 330—Three gross anatomical subdivisions of the temporal bone

construction lends itself to communication peripherally through the eustachian tube, injury to the middle ear with a rupture of the tympanic membrane further predisposes the temporal bone to infection The most frequent traumas

of the temporal bone lie in the field of otology and I, therefore, shall treat the anatomic classification from an otologic rather than from a general neurosurgical standpoint

The gross anatomical considerations are as follows (1) *the squamosa*, (2) *the mastoid*, and (3) *the petrosa* (See Fig 330)

The detailed components are, briefly, these

- 1 External auditory canal
- 2 Temporomandibular joint
- 3 Zygoma
- 4 Tympanic membrane
- 5 Middle ear
- 6 Ossicles { malleus
incus
stapes
- 7 Eustachian tube
- 8 Mastoid antrum
- 9 Labyrinth { acoustic { cochlear
static { vestibular
(or kinetic) { osseous } fluid { perilymph
membranous } endolymph
- 10 Vestibule
- 11 Windows { round
oval
- 12 Facial nerve { horizontal
vertical
- 13 Styloid process
- 14 Muscles { stapedius
tensor tympani
- 15 Internal auditory meatus
- 16 Carotid canal
- 17 Petrous apex

The intimate nerves involved are

- 1 Auditory or 8th nerve
Cochlear and vestibular
- 2 Facial or 7th nerve
Pars intermedia of Wrisburg
Chorda tympani
Geniculate ganglion
- 3 Abducens or 6th nerve
- 4 Trigeminal or 5th nerve
Gasserian ganglion (3 branches)

The major blood vessels involved are briefly these

- 1 The middle meningeal artery
- 2 The temporal artery
- 3 The lateral sinus
- 4 The superior petrosal sinus
- 5 The jugular bulb
- 6 Carotid plexus of the middle ear
- 7 Carotid canal
- 8 Internal carotid artery

Pathology.—Injury to the brain tissue, particularly injury involving the brain stem and the region of the floor of the fourth ventricle where

it can damage the cochlear and vestibular nuclei and their central connections, is of special interest to the otologist. The type of injury includes that of contusion, petecbial hemorrhage, tearing of blood vessels, laceration of cranial nerves, and damage to the vasomotor center and vasomotor nerves resulting in irritation of the vestibular nuclei. There may also be injury to the cochlear and vestibular end-organs. Here a transverse fracture of the otic capsule would destroy the sensory epithelium, a longitudinal fracture does not usually invade the capsule but is associated with bleeding of the basal coil of the cochlea and involves the eighth nerve at the internal auditory meatus interfering with the movements of perilymph and endolymph and with the entire pyramid (See Fig 327)

Symptoms.—Symptoms vary with the structures involved, and may be limited to various parts of the temporal bone in conjunction with skull fractures with or without brain injury. Although some fractures are asymptomatic, it is reasonable to assume that with more structures involved there will naturally be more obvious reactions and more pronounced symptoms. And, since the most frequent symptoms of head injury, other than headache, lie in the field of otology, I shall build the symptomatology around that premise.

Examination of the patient should determine the following (1) the presence or absence of consciousness, (2) the presence or absence of strabismus, (3) the condition of the pupils and the pupillary reflexes, (4) the presence or absence of blood or cerebrospinal fluid in the external auditory canal, condition of the drum, or the presence of a hematotympanum, as revealed upon otoscopic examination, (5) the study of the cranial nerves, (6) the history of the cochlear or vestibular apparatus prior to injury, and (7) a study of the vestibular mechanism in its relationship to the temporal bone injury.

Local Symptoms.—Fresh blood may be found or the escape of cerebrospinal fluid may be noted in the external auditory canal. Direct bleeding from the mouth, nose, or ears may indicate a basal fracture. Bleeding in some cases may be delayed for several days or for a period of a week or so. Hemorrhage is present when there is damage to the external auditory canal, perforation of the ear drum, or injury to the labyrinth and cochlea. Edema and ecchym-

osis in the region of the mastoid process may be present and often occur after the third or fourth day. This is known as Battle's sign and should be watched carefully as there may be an injury to the large blood vessels or the cranial sinuses. Subjectively the patient may complain of tinnitus, impairment in hearing, and dizziness.

Vestibular and labyrinthine symptoms consist in vertigo, ataxia, and nystagmus. When nystagmus is present, soon after the injury it is toward the injured side owing to overactivity of the normal ear, it is horizontal and rotatory in character. There is a quick component to the good side and a slow component to the injured side. The patient lies on the uninjured side in a flexed or curled up position. He looks in the direction of the injured side or slow component.

If impairment in hearing develops it may be conductive or perceptive. The hearing may be tested by voice, tuning forks, watch, and/or audiometer. Hearing tests will disclose a conductive deafness when there is only an involvement of the middle ear and tympanic membrane, perceptive deafness will be noted when there is damage to the osseous or membranous labyrinth.

When there is a complaint of vertigo, a spontaneous nystagmus, horizontal and rotatory in nature, may be noted.

Barany's tests will disclose the following: (1) Rotation will reveal hypersensitivity at first, and later hyposensitivity and complete loss of response when there is ablation of the labyrinth. (2) The Romberg and past pointing tests are positive. Falling and past pointing are positive to the side involved.

Facial palsy is noted when there is injury to the seventh nerve.

Hyperesthesia and hypesthesia are due to injury at the petrous apex in the region of the gasserian ganglion.

There may be a homolateral drooping of the lower face when the motor portion of the fifth nerve is involved.

Objective Symptoms—The examining physician should look for edema and suggestion of mastoid area. He should determine the status of external auditory canal, noting the presence or absence of edema, ulceration, fresh blood, and inspissated blood. The condition of the temporomandibular joint, as to motility, trismus, dislocation, or the presence of fracture or deformity, should be ascertained. In exami-

nation of the tympanic membrane the following should be noted: (1) intact or perforated tympanic membrane, (2) normal or abnormal landmarks, (3) discoloration, (4) fluid level or hairline, (5) hematotympanum, (6) bleeding, (7) escape of cerebrospinal fluid, (8) presence or absence of pus (otitis media), (9) position or dislocation of ossicles if the tympanic membrane is destroyed.

Diagnosis—In all cases of fracture or suspected fracture of the temporal bone, otoscopic examination should be made as promptly as possible. In the event that there is bleeding, one should not disturb the blood clot. At first it will be difficult to determine whether the escaping fluid is blood or a mixture of blood and cerebrospinal fluid. In either case disturbance of the blood clot or the ear should be very carefully avoided.

When there is injury to the squamous portion of the temporal bone it may involve either the temporal or middle meningeal artery. When the temporal artery is injured there will be edema in the region of the squamous portion of the temporal bone spreading forward, backward, and upward, depending upon the extent of the injury and the quantity of blood accumulated in that area. The external portion of the skin is generally discolored and ecchymotic, and upon palpation there will be pitting on pressure. Injury to the middle meningeal artery is quite serious depending upon the quantity of blood accumulated epidurally. When a great deal of blood accumulates pressure symptoms develop and are characterized by unconsciousness, contralateral motor palsy, Jacksonian convulsions, and increased intracranial pressure.

The longitudinal fractures are not as destructive as the transverse. The structures involved are usually the external auditory canal, temporomandibular joint, and middle ear and its contents. The symptom complex here includes hemorrhage from the ear, contusion and sugillation of the tissues in the external auditory canal, rupture of the tympanic membrane, and the escape of the cerebrospinal fluid. Should the fracture involve the epitympanum there may also be injury to the ossicles, with occasional facial palsy. Fracture through the mastoid will produce edema, ecchymosis, and sugillation, and if it extends into the region of the antrum it may cause facial palsy. A great deal of bleeding may occur because of injury to the lateral

sinus superior petrosal sinus and jugular bulb

Fracture through the petrous portion of the temporal bone is extremely serious in nature and the pathologic anatomy and symptomatology is usually bizarre in nature due to the fact that so many important structures have become affected. The most prevalent symptoms in this type of fracture are (1) the collection of fluid in the middle ear without rupture of the tympanic membrane (2) bleeding from the ear with rupture of the tympanic membrane and (3) escape of cerebrospinal fluid through the middle ear. The two important nerves that frequently become involved are the seventh and eighth. When the seventh nerve is involved there is homonymous paralysis of the face and loss of gustatory sense due to injury of the chorda tympani. There may also be neuralgic pains in the distribution of the nerve of Wrisberg. When the eighth nerve is involved the cochlear or vestibular portion may be affected. When the cochlear portion is involved there is complete deafness of the perceptive type and from a medicolegal standpoint the deafness is serious, profound and permanent. This type of deafness is generally due to a collection of blood in the labyrinth or perilymph or to damage to the cochlea. The involvement of the vestibular portion of the eighth nerve will produce vertigo which is usually subjective and may be intermittent or continuous. There is a nystagmus which is rotatory and horizontal in nature with a slow and fast component. When a vertical nystagmus appears it is usually supratentorial and is not due to labyrinthine damage. There is also past pointing and a positive Romberg reaction. In the event that the patient is unconscious an external deviation of the eyes to the side of the injury may be noted. Injury through the internal auditory meatus involving the seventh and eighth nerves produces vestibular as well as cochlear symptoms and also involves the geniculate ganglion. One is apt to notice a herpes acoustica due to the injury of this ganglion and also a facial palsy.

Injury at the petrous apex in the region of the canal of Dorello may produce an external rectus palsy. Injury at the petrous apex in the region of the gasserian ganglion will produce trifacial neuralgia, hyperesthesia, hypesthesia or complete anesthesia in one or all of the three branches. There may also be paralysis of the lower facial muscles due to involvement of the

motor portion of the trigeminal nerve. Fracture in the region of the eustachian tube and carotid canal may produce bleeding which may require ligation of the internal carotid artery. At times the osseous part of the carotid canal may help to stop the hemorrhage spontaneously and no other mechanical means will be necessary. But the blood clot remaining will exert pressure upon the sympathetic nerves supplying the carotid artery. Under these circumstances a Horner's syndrome may occur producing a myosis and drooping of the upper eyelid.

One of the pertinent medicolegal problems confronting us is an injury occurring through the labyrinthine capsule where the otic capsule does not always heal by callus formation. Healing takes place by fibroconnective tissue and remains so for many years or for a lifetime. This peculiar morphologic anomaly in faulty healing creates a communication with the periphery through the eustachian tube. Latent meningitis is a factor frequently encountered in such injuries. There are cases on record where fifteen or twenty years have elapsed between the injury and meningitis and the root of infection was through the injury of the otic capsule which never healed by callus formation.

Longitudinal or transverse fractures commonly lead to facial paralysis. Nerve injury is caused by the stretching and tearing of the nerves in the region of the internal auditory meatus or by hemorrhage into the fallopian canal. Facial paralysis may develop immediately or may be delayed for a period of several weeks and such paralysis may be complete or partial. Reaction to degeneration will determine the viability of the nerve. When it is supranuclear there is no reaction to degeneration. When it is infranuclear or peripheral then the reaction of degeneration is positive.

Vestibular Examination—The diagnosis of a typical unilateral labyrinthine fracture by vestibular examination is shown in table on page 413.

CONCLUSIONS OF VESTIBULAR EXAMINATION OF TYPICAL UNILATERAL CASE OF LABYRINTHINE FRACTURE

1. There is a spontaneous mixed nystagmus (rotatory and horizontal) to the right upon right lateral gaze. The patient falls to left in Romberg's position and falls forward with the head turned to the right and backward with head turned to the left. Also falls to the left upon pelvic girdle movement tests. Past points with both arms markedly to the left.

2. In the turning tests the duration of nystagmus is markedly reduced (2/3) when the patient is turned to

VESTIBULAR EXAMINATION
Typical Unilateral Labyrinthine Fracture

Name	H C	Age		Date	
Address	Referred by				
Case History	Recent destruction of left labyrinth as a result of injury				
Symptoms	Dizziness	~	none		
	Tinnitus	~	in left ear		
	Deafness	~	total in the left ear		
	Staggering	~	to the left		
<hr/>					
Spontaneous Phenomena					
Nystagmus				Past	Pointing
Looking right		Romberg	- to left	Right Arm	Left Arm
Looking left		Turning head to right	- falls forward	2' to L	2' to L
Looking up	- none	Turning head to left	- falls backward		
Looking down	"	Pelvic girdle movements	- falls to left		
Looking ahead	"				
<hr/>					
Turning Tests					
Nystagmus		Vertigo		Past	Pointing
To right				Right Arm	Left Arm
Duration	- 8 sec	Falling to R, very mild		3" to R	4' to R
Amplitude	- med med	8 seconds			
To left					
Duration	- 16 sec	Falling to L very mild		8 to L	6' to L
Amplitude	- med med	16 seconds			
<hr/>					
Caloric Tests					
Right Ear					
Verticals		seconds		6" to R	8' to R
After 30 sec					
Amp	med med			8' to R	8' to R
Head back					
Amp	med med				
Left Ear - none		seconds			
Verticals				2" to L	2 to L
After 5 min					
Amp				2 to L	2' to L
Head back - none				same -	spontaneous
Amp					
Ears A D - normal					
A S - deaf					

the right and moderately reduced (1/3) when turned to the left. The duration of vertigo corresponds to duration of nystagmus. Falling is very mild bilaterally and especially so when turned to the right. Past pointing is decreased bilaterally in about the same proportion as the duration of the nystagmus and vertigo.

3 In the caloric tests the right vertical and right horizontal canals give prompt and good response. The left labyrinth shows that the vertical and the horizontal canals give no response insofar as the nystagmus and past pointing are concerned.

4 The patient may or may not be susceptible to ear stimulation.

5 Hearing in the right ear is normal and totally ab-

sent in the left ear. Variations may occur in various responses depending upon compensation phenomena. Following division of one vestibular nerve or destruction of outer labyrinth by inflammation, the vestibular reaction of the other ear may be temporarily or permanently lost without any changes in the cochlear function in the normal ear.

Differential Diagnosis in Labyrinthine Injury
—The points in the differentiation between labyrinthine injury, cerebellopontile angle tumor, and Ménière's disease are presented in the following table.

sinus, superior petrosal sinus, and jugular bulb

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2. In the turn of the head tests the duration of nystagmus is markedly reduced (2/3) when the patient is turned to

VESTIBULAR EXAMINATION

Typical Unilateral Labyrinthine Fracture

Name	H C	Age	Date
Address		Referred by	
Case History Recent destruction of left labyrinth as a result of injury			
Symptoms	Dizziness - none Tinnitus - in left ear Deafness - total in the left ear Staggering - to the left		
Spontaneous Phenomena			
Nystagm is		Past	Pointing
Looking right	Romberg - to left	Right Arm	Left Arm
Looking left	Turning head to right - falls forward	2 to L	2 to L
Looking up - none	Turning head to left - falls backward		
Looking down	Pelvic girdle movements - falls to left		
Looking ahead			
Turning Tests			
Nystagmus	Vertigo	Past	Pointing
To right		Right Arm	Left Arm
Duration ~ 8 sec	Falling to R very mild	3 to R	4 to R
Amplitude - med med	8 seconds		
To left			
Duration - 16 sec	Falling to L very mild	8 to L	6 to L
Amplitude - med med	16 seconds		
Caloric Tests			
Right Ear			
Verticals	seconds	6 to R	8 to R
After 30 sec			
Amp med med		8 to R	8 to R
Head back			
Amp med med			
Left Ear - none	seconds		
Verticals		2 to L	2 to L
After 5 min			
Amp			
Head back - none		2 to L	2 to L
Amp		same - -	spontaneous
Ears A D - normal			ous
A S - deaf			

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Differential Diagnosis in Labyrinthine Injury

—The points in the differentiation between labyrinthine injury, cerebellopontile angle tumor, and Menière's disease are presented in the following table.

DIFFERENTIAL DIAGNOSIS

	Labyrinthine Injury	Cerebellopontile Angle Tumor	Meniere Disease
History	Injury	No injury	No injury
Onset	Sudden	Gradual	Recurrent
Symptoms	Vertigo (mild or severe)	Vertigo (severe)	Vertigo (severe)
Tinnitus	Unilateral	Always unilateral	Usually unilateral
Deafness	Conductive and perceptive	Complete or perceptive	Subtotal deafness with each attack
Vestibular Reaction (Bárány's test)	Early—hyperactive Late—hypoactive or no response	Syndrome of angle lesion Early—hyperactive	No definite localization
Ocular pressure	Negative	Increased ocular pressure	Negative
Anesthesia	Negative	Anesthesia of cornea	Negative

Complications—Complications of fracture of the temporal bone include (1) acute otitis media, (2) chronic suppurative otitis media, (3) mastoiditis, (4) meningitis, (5) septicemia (6) fractures through the otic capsule with failure to heal by formation of bony callus (this may be a latent or a potential source of meningitis), (7) deafness (conductive or perceptive), (8) recurrent headache, (9) tinnitus, and (10) vertigo (when the injured labyrinth is not completely obliterated or when the normal labyrinth fails to compensate).

Treatment—It is of utmost importance to see the patient promptly after injury. The first consideration is treatment of shock. General therapy can be summarized as follows:

1 In the event of increased intracranial pressure, limitation of fluid is essential. The patient should be given magnesium sulfate and intravenous injections of glucose.

2 No opiates should be administered as such medication tends to increase cerebral edema.

3 Increased intracranial pressure signifies the accumulation of blood, possibly necessitating spinal puncture or repeated punctures, or craniotomy.

4 When there is bleeding or a mixture of blood and cerebrospinal fluid from the ear, neither the blood clot nor the middle ear should be disturbed. A piece of sterile cotton should be placed in the external auditory canal and

should be removed and changed at intervals when saturated. This should be done under strictest aseptic precautions, as manipulation may lead to infection.

5 When an acute suppurative otitis media becomes evident, one should treat the condition as such and employ the dry treatment: cotton swab, aspiration or suction with cannula, dusting powder (sulfa). Wet treatment, such as irrigation and instillation of drops, should be avoided.

6 When a suppurative otitis media becomes complicated by developing into mastoiditis, simple mastoidectomy may be performed.

7 Fracture of the otic capsule in chronic suppurative otitis media should be treated symptomatically. In the event that the suppuration does not subside, and there is evidence of intracranial complications, tympanomastoidectomy may be indicated.

8 If facial paralysis develops, faradic and short-wave stimulation and massage may be indicated.

9 When there is an injury to the facial nerve with a break in the continuity of the nerve, facial nerve plasty should be performed. This is done either by direct end-to-end approximation or by the insertion of the anterior cutaneous nerve between the distal and proximal portions.

10 Decompression of the fallopian canal

permits the escape of fluid and thus allows the edema of the nerve to subside

11 When bleeding is profuse and continuous, the source should be investigated. If bleeding emanates from the dural sinuses, it should be obliterated by packing. If the bleeding comes from an artery, it should be ligated and the clot evacuated. In bleeding from the middle meningeal artery, when a quantity of blood has accumulated epidurally, decompression is essential and evacuation of the blood clot and the checking of the hemorrhage are necessary. In fracture of the mastoid, when hemorrhage is due to injury of the lateral sinus, superior petrosal sinus, or jugular bulb, treatment consists in exposing the mastoid region, performing a mastoidectomy, and controlling the bleeding from the lateral sinus or from any of the other blood vessels by packing or other mechanical means.

12 Should a hematoma become infected it should be treated as a septic wound, by means of incision, drainage, and other supportive biochemical measures, which tend to brighten the prognosis.

13 Penicillin and other modern biological drugs may supplement the armamentarium.

Prognosis—The immediate mortality from basal skull fractures is high, from temporal bone fractures is not very high. Late meningitis mortality is fairly low. Patients having blood or cerebrospinal discharge recover without the development of meningitis unless the otic capsule itself has been fractured.

In impairment in hearing, when there is conductive deafness the prospects for complete recovery are good, and when there is perceptive deafness the prospects for improvement in hearing are unfavorable.

In facial palsy, where there is supranuclear palsy the prognosis is unfavorable for facial nerve recovery. In peripheral injury with resultant facial palsy, recovery may take place spontaneously when the reaction to degeneration is negative, when it is positive spontaneous recovery will not occur unless the fallopian canal is decompressed or a facial nerve plasty is performed.

MATTHEW S. ERSNER

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PART IV. LARYNX AND HYPOPHARYNX

ANATOMY OF THE LARYNX

The larynx is primarily a valvular mechanism leading into the trachea which has acquired an added phonatory function later in its phylogeny. It is made up of a more or less rigid framework of cartilages held together by ligaments and moved one on another by muscles. The interior of this boxlike structure is lined by mucous membrane which is continuous above with the mucous membrane of the pharynx and below with that of the trachea. The general shape is roughly triangular in its upper portion tapering toward a narrower cylindrical portion below.

The larynx is separated from the fourth, fifth, and sixth cervical vertebral bodies by the laryngeal portion of the pharynx, the anterior wall of which it forms. The larynx can be located readily by inspection and palpation of the anterior aspect of the neck where its thyroid cartilage produces a variable protrusion, the *laryngeal prominence* or Adam's apple.

Laryngeal Cartilages and Their Articulations—The cartilages which are of most significance in the valvular and phonatory activities of the larynx are the "shieldlike" *thyroid cartilage*, the "ringlike" *cricoid cartilage*, and the right and left *arytenoid cartilages*. These cartilages are shown in their natural relation to each other in Figure 331 in which their important described parts are labeled. It should be noted that each inferior cornu of the thyroid cartilage articulates with an articular facet on the cricoid near where the lamina and arch meet. These joints, which are synovial or true joints, allow the cricoid cartilage to be tilted in relation to the thyroid cartilage. Each arytenoid cartilage articulates by means of a synovial joint with a roughly half cylindrical articular surface on the superolateral aspect of the lamina of the cricoid, the long axis of which is about halfway between vertical and horizontal. The articular surface on the inferior aspect of the muscular process of the arytenoid is somewhat reciprocal to that on the cricoid but the concavity is less than the convexity of the cricoid articular sur-

face. The crico arytenoid joint allows the arytenoid cartilage to slide toward and away from the one of the other side and to undergo a sweeping movement which carries the vocal process toward or away from the median plane.

In addition to the above cartilages, there are also the *cartilage of the epiglottis*, which is a thin, pitted sheet of cartilage conforming to the contours of the epiglottis and tapering inferiorly into a stem which is attached to the inside of the angle formed by the thyroid laminae a little below the thyroid notch, the right and left *corniculate cartilages* (of Santorini), which are attached to the apices of the arytenoid cartilages and curve posteriorly and medially, the right and left *cuneiform cartilages* (of Wisniewski), which are small rod shape pieces in the aryepiglottic folds near the corniculate cartilages, and the right and left *triticeous cartilages*, which may be present in the lateral thyrohyoid ligaments.

Beginning between the twentieth and thirtieth year of life there is usually some calcification of the laryngeal cartilages. The most common sites are the posterior and inferior parts of the thyroid laminae and the cricoid lamina but other parts also become ossified and the calcification may be quite complete in the latter decades.

The Laryngeal Cavity and Its Walls.—On each lateral wall of the laryngeal cavity (Fig. 332) there are two horizontally placed folds, the *ventricular fold* and the *vocal fold*. The vocal fold, or true vocal cord, is the inferior of the two and it runs from the vocal process of the arytenoid forward to reach the angle between the two thyroid laminae a little above halfway between the thyroid notch and the inferior border of the thyroid cartilage. The ventricular fold, or false vocal cord, is above the vocal fold and under most conditions does not project as far into the cavity of the larynx as does the vocal fold. It extends from just above the thyroid end of the vocal fold to the anterolateral aspect of the arytenoid, a little away from the vocal process. For descriptive purposes the ventricular and vocal folds are thought

of as dividing the cavity of the larynx into three parts supraglottic, glottic, and infraglottic

The supraglottic region or *vestibule of the larynx*, communicates above with the pharynx by the *entrance to the larynx* (aditus laryngis), which is bounded anteriorly by the free margin of the epiglottis posterolaterally by the aryepiglottic folds running from the lateral margins of the epiglottis to the arytenoids, and posteriorly by the mucous membrane covering the arytenoid and corniculate cartilages and running from one arytenoid to the other below the interarytenoid notch. This entrance to the larynx faces almost posteriorly. On the anterior wall

opening through which the supraglottic and glottic portions communicate with the infraglottic portion. The name *rima glottidis* is applied to this opening, a little more than the anterior half of which is bounded by the vocal folds—the *intermembranous part of the rima*. The rest is bounded by the mucous membrane covering for the most part, the arytenoid cartilages, and is called the *intercartilaginous part of the rima*.

The *infraglottic region* extends from the *rima glottidis* to the lower border of the cricoid cartilage where the cavity continues into the trachea without any very striking line of demarcation on the wall of the cavity.

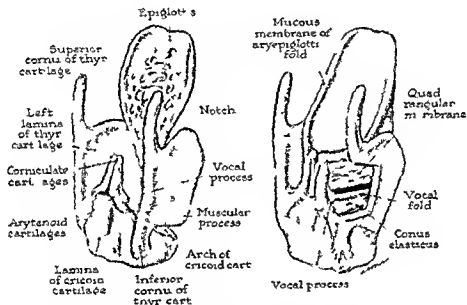


Fig. 331.—The cartilaginous and fibrous tissue framework of the larynx, viewed from behind and the right

of the vestibule is the tubercle of the epiglottis. The inferior limit of the vestibule is the free margin of the ventricular fold.

The *glottic region* is ordinarily considered to extend from the free margin of the ventricular fold to the free margin of the vocal fold, although the word *glottis* is commonly used to mean only the vocal folds and the intervening space (the sound producing structures). Between the vocal and ventricular folds of each side there is an expansion of the cavity of the larynx known as the *ventricle of the larynx* which usually undermines the ventricular fold to some extent and has a variable small diverticulum running superiorly from its ventral part, called the *appendix of the ventricle*. The narrowest part of the laryngeal cavity is the

Ligaments and Membranes—The fibrous tissue within the larynx and attaching the larynx to adjoining structures, can be demonstrated (by dissection) to be present in sheets and bands which are called 'membranes' and 'ligaments' (Fig. 331). The number of such structures which one finds depends as in the other portions of the body, in part on the way in which he divides the tissues. For descriptive purposes one can think of three groups of structures: those connecting the larynx to adjoining structures (extrinsic ligaments), the ligaments of the joints, and the fibrous framework of the larynx.

The sheet of fibrous tissue attaching below to the superior border of the laminae and the anterior borders and tips of the superior cornua

of the thyroid cartilage, and above to the superior margin of the posterior aspect, the body, and the posteromedial aspect of the greater cornua of the hyoid bone, is called the *thyrohyoid membrane*. The parts of this membrane between the superior cornua of the thyroid and greater cornua of the hyoid are to some extent thickened and may be referred to as *lateral thyrohyoid ligaments*. The *cricotracheal ligament* connects the cricoid cartilage with the first tracheal ring, running from the inferior border of the former to the superior border of the latter.

The cricothyroid and crico-arytenoid joints, being synovial or true joints, have fibrous capsules in which one can make out certain thickenings, or ligaments, but these do not merit

the aryepiglottic and ventricular folds, and its margin in the ventricular fold is sometimes described as a ventricular ligament. The lower portion of the elastic membrane, or the *conus elasticus*, is attached below to the entire length of the superior margin of the arch of the cricoid cartilage from one arytenoid articulation to the other. Above it attaches anteriorly to the thyroid cartilage from the inferior margin at the angle between the laminae up to the attachment of the vocal fold, and posteriorly to the inferior border of the vocal processes of the arytenoid cartilages. Between these two attachments the conus elasticus has free margins in the vocal folds which are thickened to form the vocal ligaments. The portion of the conus elasticus near the midline anteriorly and attached above to the inferior margin of the thyroid cartilage is thicker and stronger than the rest and may be given a special name, the *median cricothyroid ligament*.

Intrinsic Muscles of the Larynx—The intrinsic musculature of the larynx (Fig 333) which is most important in the valvular and phonatory activities of the larynx is that which acts on or produces movements at the cricothyroid joint and that which functions at the crico-arytenoid joint. These muscles are described in the succeeding paragraphs. There is, in addition to these two groups, some musculature in the aryepiglottic folds which is neither as well defined nor as well developed and is more in the nature of scattered fasciculi. However, two hands have been found frequently enough so that they have been given special names—the *thyro-epiglottic muscle* and the *aryepiglottic muscle*, both of which are running toward the side of the epiglottis, the former from the thyroid and the latter from the arytenoid cartilage. They are partially blended with the thyro-arytenoid muscle. This musculature in the aryepiglottic folds is usually interpreted as an imperfect sphincter of the opening of the larynx.

Muscles Acting on the Crico-arytenoid Joint

—The *posterior crico-arytenoid muscle* arises from the homolateral half of the posterior surface of the lamina of the cricoid cartilage and inserts into the posterior aspect of the muscular process of the arytenoid cartilage. It pulls the muscular process downward and medially which causes the vocal process to be swept laterally and a little superiorly. Probably also, in conjunction with the lateral crico-arytenoid muscle

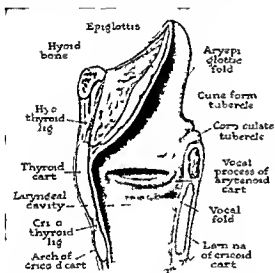


Fig 332—The larynx as seen in midsagittal section

individual mention. There is, however, one accessory ligament of the crico-arytenoid joint which runs from the posteromedial aspect of the base of the arytenoid cartilage to a little to one side of the midpoint of the superior margin of the lamina of the cricoid, the *posterior crico-arytenoid ligament*, which is functionally important in that it pulls the arytenoid posteriorly when the superior margin of the cricoid moves posteriorly as the cricoid is tilted and it causes the cricoid to be tilted in the opposite direction when the arytenoid is pulled forward.

The fibrous framework of the larynx or the *elastic membrane of the larynx* is blended with the deeper layers of the mucous membrane lining the larynx. Its upper part, which often is not well defined, helps form the framework of

its lateral fibers would slide the arytenoid away from the midline

The *lateral crico arytenoid muscle* arises from the posterior half or so of the superior border of the arch of the cricoid cartilage and is inserted into the anterolateral surface of the muscular process of the arytenoid cartilage. This muscle, acting with the thyro arytenoid, causes the vocal process of the arytenoid to be swept medially and, as has been stated, probably helps part of the posterior crico arytenoid muscle in sliding the arytenoid laterally

The *thyro arytenoid muscle* arises from the inner aspect of the lower half of the angle formed by the laminae of the thyroid cartilage and inserts into the lower part of the anterolateral

The *arytenoid muscle* is unpaired. It runs from one arytenoid cartilage to the other being attached to a little more than the lower half of the posterolateral border of the cartilage and partly on the medial aspect of the muscular process. Most descriptions mention a transverse portion, which is the main bulk of the muscle, and an oblique portion in the form of an "X," superficial to the transverse part. Frequently the oblique portion is poorly defined. The arytenoid muscle slides and, probably also, tilts the arytenoid cartilages toward each other.

Muscles Acting on the Cricothyroid Joint—The *cricothyroid muscle* arises from the anterolateral aspect of the arch of the cricoid cartilage and inserts into the anterior border of

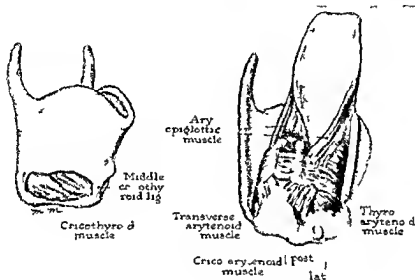


Fig. 333—The intrinsic muscles of the larynx

aspect of the arytenoid cartilage from the tip of the vocal process back onto the muscular process. In spite of the fact that in dissection the separation is not obvious, the medial or internal part of this muscle, which is adjacent to the vocal ligament, is usually given a special name, the *vocal muscle*. The thyro-arytenoid muscle as a whole, as has been stated previously, helps the lateral crico arytenoid muscle to cause the vocal process of the arytenoid cartilage to be swept medially. In addition it has an indirect action on the cricothyroid joint which will be discussed presently. The vocal muscle is described by some as sending fibers medially to end on the vocal ligament and thus in some fashion being able to make firm or stabilize separate portions of the vocal fold.

the inferior cornu and the adjacent part of the inferior border of the lamina of the thyroid cartilage. It elevates the arch of the cricoid cartilage thus tilting the lamina posteriorly.

The thyro arytenoid muscle (described previously) has an indirect action on the cricothyroid joint in that it pulls the arytenoid cartilage forward and that in turn, by means of the posterior crico-arytenoid ligament, pulls the superior part of the lamina of the cricoid forward thus producing an action at the cricothyroid joint opposite to that of the cricothyroid muscle.

Analysis of the Mechanism of the Larynx—The entire mechanism of the larynx is quite complicated but the following account is an attempt to show how the previously described

structures enter into the activities of the larynx which are rather arbitrarily split up in this discussion for the purpose of simplification

Phonatory Activity—For phonation the vocal folds are approximated or adducted. This is accomplished by the thyro arytenoid and lateral crico arytenoid muscles which, as has been stated, cause the vocal processes of the arytenoid cartilages (to which the vocal folds are attached) to be swept medially. The tension of the vocal folds, which probably is related to the pitch of the sound, depends on the distance between the two attachments of the vocal folds (the vocal processes of the arytenoids and the angle between the thyroid laminae) and that distance is increased (tightening the folds) by the crico thyroid muscles which tilt the cricoid posteriorly and thus pull the arytenoids posteriorly by means of the posterior crico arytenoid ligaments. The distance between the anterior and posterior attachments of the vocal folds is shortened (loosening the folds) by the thyro arytenoid muscles pulling the arytenoid cartilages forward (p 423)

Respiratory Activity—In respiration the vocal folds are held apart or partially abducted by the posterior crico arytenoid muscles which as has been stated, cause the vocal processes of the arytenoid cartilages to be swept laterally. The intercartilaginous part of the rima glottidis is held open by the arytenoid cartilages being held laterally by part of the posterior crico arytenoid and probably the lateral crico arytenoid muscles. The lamina of the cricoid cartilage is kept from tilting forward and thus shortening the rima by the crico thyroid muscles. In forced respiration the diameters of the rima can be increased by more forceful activity of the muscles which have previously been described.

Valvular Activity—**INLET VALVE**—In prevention of the entrance of foreign material into the trachea, the entrance to the larynx (laryngeal aditus) is closed and the rima glottidis is closed. Probably these two actions take place simultaneously, as a rule. The entrance to the larynx is closed by several actions: (1) the elevation of the larynx by extrinsic muscles which brings the structures below up against the epiglottis, (2) the arytenoid cartilages brought forward by the thyro arytenoid muscles (cricoid tilting forward at the cricothyroid joint), (3) the arytenoid cartilages brought together by the arytenoid muscle, (4) sphincter action of the musculature in the aryepiglottic folds. The rima

glottidis is closed by the adduction of the vocal folds and the approximation of the arytenoid cartilages both of which have been discussed.

OUTLET VALVE—The ventricular folds act as a valve which can prevent the escape of air from the trachea and lungs and thus make it possible for the intrathoracic pressure to be built up for many purposes. The ventricular folds are approximated by the arytenoid muscle, which approximates the arytenoid cartilages and tilts them toward each other, and probably also by the thyro arytenoids and perhaps the lateral crico arytenoids. It is likely that once the ventricular folds are approximated they act as a mechanical valve, owing to the air getting into the ventricle, until the arytenoid cartilages are separated by muscular pull.

Nerve Supply—As the superior laryngeal branch of the vagus nerve is approaching the larynx from above it divides into an external branch, which goes to supply the cricothyroid muscle and an internal branch, which pierces the thyrohyoid membrane to get into the interior of the larynx and to spread out to the mucous membrane of the larynx giving afferent supply and efferent supply to the glands contained therein. This branch probably gives some supply to the arytenoid muscle. The rest of the intrinsic musculature of the larynx is supplied by the recurrent or inferior laryngeal branch of the vagus nerve (p 424). There are probably fibers from the cervical chain ganglia for thoracolumbar or sympathetic supply of the glands in the mucous membrane which come by way of periarterial plexuses. There are taste buds on the posterior surface of the epiglottis.

Blood Supply—The principal arteries supplying the larynx are the superior laryngeal branch of the superior thyroid branch of the external carotid, and the inferior laryngeal branch of the inferior thyroid branch of the thyrocervical branch of the subclavian. The superior laryngeal accompanies the internal branch of the superior laryngeal nerve through the thyrohyoid membrane and in its spread on the interior of the larynx. The inferior laryngeal artery accompanies the inferior laryngeal nerve. There are a few small branches of the cricothyroid branch of the superior thyroid which enter the larynx. The veins accompany the arteries as far as the superior and inferior thyroid veins, the former of which empties into the internal jugular vein and the latter usually into the left innominate vein.

Lymphatic Drainage—The lymphatics of parts of the larynx other than the mucous membrane are not well known. The lymphatic capillary network in the mucous membrane of the supraglottic region is more or less separated from that of the infraglottic region by the scarcity of network at the margins of the vocal folds. The supraglottic network is drained primarily by vessels which accompany the superior laryngeal blood vessels and empty into nodes of the superior deep cervical group. The infraglottic network drains mostly posteriorly to get out of the larynx, and then to lower nodes of the superior deep cervical group.

Microscopic Anatomy—The mucous membrane of the larynx consists of an epithelium which is ciliated pseudostratified columnar except in the region of the vocal folds and the anterior surface of the epiglottis where it is stratified squamous and a lamina propria composed of fibrous connective tissue with many elastic fibers and containing a variable number of lymphocytes. The submucosa contains tubuloalveolar glands, mixed in type except in the vocal folds.

The cartilages of the larynx are all hyaline with the exception of the cartilage of the epiglottis, the corniculate and cuneiform cartilages and the apices and vocal processes of the arytenoid cartilages which are yellow elastic.

The intrinsic muscles of the larynx are voluntary striated.

JOHN FRANKLIN HUBER

PHYSIOLOGY OF THE LARYNX

Phylogeny—All animal life originally was aquatic, and derived oxygen from water. Therefore lungs were unnecessary. When fishes were stranded by receding waters gills could not supply oxygen and survival depended upon evolution of lungs for this purpose. To protect the lungs from intrusion of mud or other solid substance a closing mechanism was required, and a crude equivalent of a larynx was evolved for this purpose. Existing species are of the subclass of fishes, *Dipnoi* which have lungs and a pulmonary circulation for breathing air, yet retain their gills for obtaining oxygen from water. Somewhat higher forms of vertebrates have lost the gills and developed better lungs with the

laryngeal air valve to keep out solid matter and they have acquired a check valve to retain air gulped or swallowed. Thus was evolved an air valve with an opening and a closing mechanism. This valvular mechanism was concerned with respiration, not phonation. To the protective and the respiratory functions the phonatory was added incidentally.

Consideration of one of the phylogenetically early vertebrates will illustrate how this could have occurred. The frog will serve as an example.

When a frog sitting on the bank of a pond is surprised by the sudden approach of a person it precipitately gulps a supply of air with a sound resembling a snap as it plunges into the water to escape. The sound is due chiefly to the snapping shut of the mouth, probably also in part to the closing of the valve-like orifice of the simple passage to the lungs. The sudden and powerful general muscular contraction exerted in a great leap contributes largely to the sudden shutting that produces the sound. It is not made when the frog enters the water without the element of alarm and escape. It is not intentionally made, but here we have a very primitive type of phonation associated with a valve-like closure at the top of the airway that is incidental to escape.

Coming to the highest phonatory species *Homo sapiens*, or man, we note that the first intentional sound made by a baby is a repetition of a sound such as a hiss that he has discovered accidentally. The newborn baby's cry is entirely unintentional, but when his intelligence develops he uses the cry to call his mother, and ultimately to arouse her sympathy and to induce her to yield to his wants.

These simple examples illustrate in an elementary way the huge mass of biologic data supporting the fundamental fact that the primary vital function of the larynx was and still is protective and respiratory, whereas the phonatory function was secondary and incidental, and probably first occurred unintentionally. Phylogenetic phases of other laryngeal functions will be mentioned in subsequent paragraphs.

Through the ages the simple form of larynx possessed by the lower vertebrates has evolved into the marvellous laryngeal mechanism of man with its nine known functions, climaxed by highly elaborated use of the phonatory function for intelligent speech. So impressive is this phonatory function that we are all prone to overlook the older and vitally more important functions.

Functions of the Larynx—The nine human functions of the larynx in man are the following (1) protective, (2) respiratory, (3) circulatory, (4) fixative, (5) deglutitory, (6) tussive, (7) expectorative, (8) phonatory, and (9) emotional

Protective Function—This function, vitally necessary for all forms of animal life having lungs, has three phases (1) by closure of the airway, swallowing of food is permitted without intrusion of the food, (2) by closure of the larynx, accidental intrusion of any other substance is prevented, (3) by cooperation with the cough reflex, any intruding substance that has passed the first line of pulmonary defense is promptly expelled. The epiglottis participates in the protective function of the larynx by deflecting food and foreign bodies from the laryngeal orifice

Respiratory Function—The passive role as air channel is but a minor part of the laryngeal contribution to respiration. It participates mechanically and biochemically in the regulation of the interchange of carbon dioxide tension of the blood and in maintaining the acid base balance in the blood and tissues. This is done by the sensitive nicety of valvular control of the area of cross section of the glottis

Circulatory Functions—In addition to the biochemical effects on the circulating blood mentioned in the preceding paragraph, the alternating plus and minus pressures of the air content of the tracheobronchial tree and pulmonary parenchyma exert a pumping action on the blood circulating in the thin and resilient walls of the air vesicles, and in some degree on the heart itself

Fixative Function—The thorax requires fixation for two purposes (1) to give support to powerful voluntary muscular use of the arms and (2) to give support to strong voluntary action of the muscles of the abdominal wall, as at stool and in parturition. In phylogenetic sequence the larynx had developed great power of valvular closure before quadrupeds, developing powerful forelimbs, required this fixative assistance, for example, when springing upon their prey. Later, life in trees called for thoracic fixation for suspension and prehension by the forelimbs. In man, powerful exertion such as lifting is always immediately preceded by laryngeal closure to fix the thorax partly filled with air. A patient breathing through the neck after tracheotomy or laryngectomy is hand-

capped in heavy lifting for want of this power of thoracic fixation. Wearing a valvular cannula will partially help him

Deglutitory Function—There are three mechanical elements in laryngeal assistance in deglutition (1) In swallowing, the inferior pharyngeal constrictors, arising from both the cricoid and thyroid cartilages, draw the larynx upward to grasp the bolus and force it downward (2) The larynx closes to prevent entrance of food or drink into the airway by contraction of its orifice and coverage by the epiglottis. The usual belittling of the epiglottis as a protective factor in deglutition we believe is an error. By capping the closed laryngeal orifice it prevents lodgment of food particles in the cuplike closed orifice and also makes a more perfect closure (3) The epiglottis, shaped like two miniature plows with flat sides together, forms two mold boards, a right and left, that split the bolus and pass the halves into the right and left pyriform sinuses respectively. It is true that the epiglottis may be amputated, but the choking and strangling are usually severe until vicarious adjustment is made

Tussive and Expectorative Functions—These are protective functions also, they constitute a second line of defense in case any foreign substance has succeeded in passing the closing glottic first line. Additionally they cooperate in expelling what may be called "endogenous" foreign bodies, secretions, sequestra, or bacterial accumulations, and, occasionally, they help expel an intruder that has penetrated the thoracic wall, such as a bit of clothing carried in by a bit of coal in a mine explosion, a shell fragment, or a projectile, or, quite rarely, even the missile itself may be expelled through natural passages. The endogenous substances, such as pus, may originate in the trachea or larger bronchi, whence they are readily expelled by the bechic blast. In the smaller bronchi and the peripheral pulmonary parenchyma, where an air blast is feeble or impossible, the substance requiring expulsion is continuously wafted upward by ciliary action, and by the tussive squeeze² is forced upward to the larger bronchi where expulsion is completed by the bechic blast. When local disease or injury has destroyed the cilia (or drugs have paralyzed them) the powerful tussive squeeze alone is, ordinarily, adequate. At each cough the powerful thoracic muscles compress the invaded lobe, forcing out the secretion in a way comparable

to the florist's manual compression of the rubber sprinkling bulb

The laryngeal coordinating participation in the cycle resulting in the expulsion of foreign or endogenous substance is valvular. The sequence of movements is given on a subsequent page in connection with laryngeal reflexes. The bechic blast carries the foreign substance with it. Sometimes a foreign substance, or a pellet of moss-agate tracheal secretion, may be shot out of the mouth like a projectile. Ordinarily, however, secretions lodge in the pharynx and are swallowed (as in younger children) or expectorated. The blast responsible for the ejection of the foreign substance, whether the substance is swallowed or expectorated, is called *productive cough*. The ordinary "clearing of the throat," usually spelled a-h e m, constitutes a voluntary but very feeble bechic cycle. In hic-cough the inspiratory rushing column of air, caused by the convulsive action of the abdominal and diaphragmatic muscles, is suddenly arrested by the slamming shut of the "cellar door" mechanism (*q v*) of the glottis due to air pressure on the ventricular floor. The sound is due to the vibration of arrest. In mechanism it is similar to the suddenly arrested gulp of air by the frightened frog mentioned in a preceding paragraph.

Emotional Function—The larynx cooperates in sobbing, crying, shuddering, and moaning, the emotional expressions of grief, distress, and terror. The phonatory function is active in moaning, crying and also in fatigue, as in yawn ing (*q v*).

Phonatory Function—In the casual thoughts of the average human being this is the chief, usually the only, function of the larynx, yet it is not of vital importance in man, under ordinary circumstances. As a mechanism it is marvelous.

MECHANISM OF NORMAL SPEECH—The larynx and the air passages of which it forms a part constitute an air column instrument to produce sounds of varying pitch.³ These sounds are not words, and the larynx cannot produce words. For articulate speech the sounds made by the laryngeal air column instrument are molded into words by the *molds of speech*. The mechanism of sound production is well illustrated by the bugle.³

The bugle is a tube containing no valves or reeds. If the whole mouthpiece be put into one's mouth, blowing breath through the bugle will produce no sound. To produce a sound the bugle player sets the column of air

contained in the bugle in vibration by firming his lips against the mouthpiece and blowing through the chink between the firm lips. His firm lips do not set the air column in vibration like a guitar string; they cut the air column into puffs and the frequency of the puffs corresponds mathematically to the pitch produced. To produce the pitch of G 388 the lips must be set at the proper degree of firmness to produce 388 puffs per second. The beginner in bugle playing is told to relax his lips for lower notes and to firm his lips more and more for the higher and higher notes. By thus changing the firmness of the edges of his lips the bugler can get notes of the five different pitches producible on the bugle. He cannot obtain more than that because he cannot change the length of the air column in a bugle. To produce the intermediate pitches the variable firming of the lips must be supplemented by cooperative change in the length of the air column as is crudely done by telescopic sliding of the trombone.

The human supraglottic and infraglottic air column is proportionately shortened for high pitches coordinately with the increased firming of the cordal edges. Similarly to the trombonist who, in order to obtain the higher pitches, in addition to greatly increasing the firming or hardening of his lips, "overblows" (that is to say he increases the air pressure that he forces through between his lips), the soprano, in producing high notes, unconsciously increases the tracheal air pressure coordinately as she firms the cordal edges and shortens the supraglottic air column by raising the larynx, additionally she narrows the ventricles and pharynx.^{2,3} The diametric diminution of the air column obviates the need for great changes in length of air column such as necessitate the long sliding range of the trombone. This power is not possessed by any muscular instrument. The nearest approach is the organ whose selective action permits the use of a pipe that is less in diameter as well as length for a higher pitch. The deep pitch of the voice of patients with laryngoptosis demonstrates the effect of lengthening the supraglottic air column. The firming of the edges of the vocal cords to change the resiliency is produced by the counter pull of the closing mechanism against the opening mechanism of the larynx. It is, therefore, simply an adaptation of the two primary and primitive functions of the larynx, dating phylogenetically from the evolutionary period when aquatic life became amphibious. In the human larynx the opening muscles (the crico-arytaenoides postici, the cricothyroides, and the cricopharyngeus) take up the slack and give fixation to resist the closing action of the closing muscles, these orbicular muscles are the crico-arytaenoides laterales, the

arytaenoides and the thyro arytaenoides. The latter pair of muscles inserted along the edges of the vocal cords effect the firming changes in resiliency. The action of all of these muscles will be herein considered with their innervation, and again with the reciprocal rocking movement of the thyroid and cricoid cartilages in connection with laryngeal paralyses.

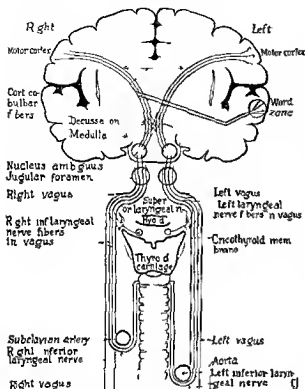


Fig. 334—Pen sketch schematically illustrating in a simplified way the fundamentals of the innervation of the larynx for performing its nine functions. For clearness intervening central structures have been omitted. The laryngeal nerves originate in the nucleus ambiguus of which there are two—one on each side of the medulla. Here are activated the automatic functions of respiratory and reflex laryngeal movements. For volitional movements the nucleus ambiguus are activated and dominated by impulses (orders) received from the cortical executive centers. These bilateral executive centers in turn receive their orders as to words from the unilateral language (or word) area located on the left side of the brain (in right handed individuals) represented diagrammatically as distributing its impulses bilaterally (see Fig. 335). To avoid impairing simplicity only of efferent pathways are indicated in this schema. It must be understood that all these pathways have their complementary afferent pathways.

From the foregoing it is clear that the control of human phonatory pitch is dependent upon three factors: (1) the controlled change in the resiliency of the vocal cords by which the number of puffs of air per second at the glottis is controlled, (2) the cooperative control of the

diameter and length of the air column in the air passages from the lips to the lungs, and (3) controlled increase in tracheal air pressure for the higher notes.

This controlled variation in pitch however, does not produce words. The vibrating phonatory air column is formed into words by the molds of speech—the pharynx, palate, tongue, teeth, and lips.

All sounds used in articulate speech, however, are not produced in the larynx, for example the hushing sound of S which is produced by the tongue pressed against the hard palate, thus narrowing the passage for the air blast to a small chink. The sound of F is produced by dental and lower labial narrowing of the passageway. Some other consonants (P, T, K) are produced by explosive release of imprisoned air. All consonants are mechanically obstructive, vowel sounds are more or less free from obstruction in the upper air-channels.

Innervation of the Larynx.—The larynx is supplied with both motor and sensory fibers (probably also with vasomotor fibers) by two pairs of nerves, on each side there is a superior and an inferior laryngeal nerve—all four of these are branches of the vagi. Both pairs of nerves carry afferent and efferent impulses, and all four nerves are connected by anastomotic or association fibers. In the distribution of these four nerves there is some overlapping as well as anastomosis and this is sometimes confusing in the diagnosis of paralysis. In health the right and left paired muscles coordinate symmetrically but in paralysis one side alone may be affected.

The *superior laryngeal nerve* is the chief vasomotor, secretory, and sensory nerve of the larynx, but it is also important as a motor nerve. After leaving the vagus (Fig. 334) it divides into two branches, one of which, the external, chiefly motor, passes downward and is distributed to the cricothyroid muscle, a few stray neurons are traceable to an anastomosis with the inferior laryngeal nerve. The other branch, chiefly sensory, enters the larynx through the thyrohyoid membrane and is distributed to the mucosa of the larynx and epiglottis. It is the afferent neurons that transmit centrally the sensation of irritation and pain in laryngeal disease. A foreign body, exogenous or endogenous, through this nerve excites the impulses transmitted centrally that come back as bilateral motor impulses to the laryngeal and thoracic

muscles, thus completing the arc known as the cough reflex. It is the watchdog of the lungs. It should be remembered that there are, additionally, afferent as well as efferent pathways in the inferior laryngeal nerves, and also a network of association fibers, central and peripheral. In addition to the just mentioned functions of the superior laryngeal nerve, this nerve is probably concerned in the excitation of the continuous tonus that maintains the glottic margins normal in contour and the glottic area of cross section proper for the circulatory balance of pulmonary air pressures. The motor function of the superior laryngeal nerve is very clear and distinct. It activates the cricothyroid muscle to rock and fix the cricoid and thyroid cartilages to resist the pull of the thyro arytenoides when these muscles firm the edges of the vocal cords to raise the pitch of the voice (Fig. 333). The inferior laryngeal nerves are the motor nerves of the musculature of the larynx (Fig. 334). They are given off by the vagus at different levels. The right inferior laryngeal leaves the vagus as the vagus crosses the right subclavian artery, under which it passes, then it ascends in the groove between the trachea and esophagus to the level of the upper border of the cricoid cartilages where it divides into two branches. The anterior branch is distributed to the crico arytenoid, the thyro arytenoid, and the arytenoid muscles. The posterior branch is distributed to the right posterior crico arytenoid muscle and to the arytenoid on both sides of the median line. The left inferior laryngeal nerve is given off by the vagus as it crosses the arch of the aorta, around which it turns to ascend and emerge from the thorax, thence upward, its position, branches, and distribution are relatively the same as those of the right inferior laryngeal nerve (Fig. 334). The inferior laryngeal nerves are independent to the extent that they can each be separately paralyzed without any detectable impairment of the other, they cooperate perfectly and synchronously though they carry impulses to groups of muscles that are directly antagonistic in action. For example, one set of muscles closes the glottis, another set supplied by the same nerve pulls the glottis open and holds it open. In phonation it is this counter pull that produces the changes in the elasticity of the cordal edges so as to yield the many changes in pitch of which the human voice is capable. In swallowing, both larynx and glottis close, but in pho-

nation the glottis closes while the laryngeal aditus is strongly held open by the opening set of muscles, the abductors.

In addition to the clearly defined motor and sensory innervation described in foregoing paragraphs there is a not so clearly understood, but definitely present, normal form of innervation known as *tonus* (Fig. 396). Tonus sometimes maintains even the paralytic vocal cord in a tonic state, when tonus is lost, as it is ultimately in many paralytic cases, the cords sag back into the relaxed position they often assume after death (see under "Bilateral Incomplete Paralysis of the Larynx"). It is probably maintained by a continuous activity in the medullary laryngeal center intimately associated with the respiratory center. The effect of the tonus is to maintain the balanced status of the vocal cords amply, yet not widely separated, in an almost motionless position during quiet respiration. The source of excitation impulses for this reflex tonus may be exerted through the superior laryngeal nerve fibers, or, possibly, it may not be in the larynx—it may be vagal. All chains of neurons are not traceable.

The Respiratory, Phonatory, and Language Centers—Generalities—It will greatly aid in acquiring a full understanding of the confusing subject of innervation of the larynx to start with the broad and general statement that there are two kinds of central executive control—one kind automatic and constant, the other more or less voluntary and intermittent. The automatic control is in the medulla, the voluntary control is in the cerebral cortex. The respiratory centers in the medulla carry on the breathing automatically, throughout life, whether the individual is asleep or awake. But, when awake, a person can voluntarily interfere with this rhythmic breathing directly, as in holding the breath, or indirectly by speaking, which starts another automatic control of breathing to coordinate the breathing with the requirements of articulate speech. A simplifying generalization is that man breathes with his medulla but when he wants to use his voice the medulla takes orders from his cortex. These orders will not be obeyed if obedience would endanger life. The individual can hold his breath but not to the point of suicide. When the carbon dioxide tension in the blood rises high enough the medulla becomes dominant and it will resume its respiratory function even before unconsciousness develops.

Before going into details, it would be helpful to grasp the rough but fundamental ideas that (1) thoughts develop in the intellectual cells of the brain, (2) to convert those thoughts into spoken words impulses go from the intellectual cells to a special one sided language zone, where (3) a set of coordinated impulses go to (a) the chest and larynx to make a sound and (b) to the lips, tongue, and palate to form that sound into spoken words

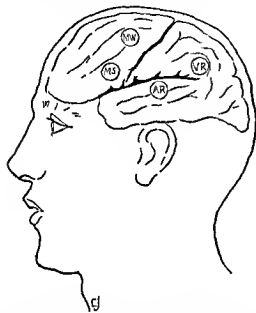


Fig 335—Schematic simplified sketch of the un lateral language ("word ") center located on the left side of the brain in congenitally right handed individuals It is here that thoughts are transformed into word impulses that are coordinately distributed through cortical executive centers (see Fig 334) to (1) the groups of muscles of the chest and larynx to produce a sound, and (2) to the groups of muscles of the pharynx, palate, tongue, and lips to mold that sound into words The language area comprises two receptive centers visual (VR) receiving word impulses from the eye and auditory (AR) receiving word impulses from the ear Two other centers are motor, one (MS) sending out impulses for spoken words, the other (MW) sending impulses to the word writing group of muscles in forearm and fingers All four centers are integrated into a language zone by intimately interwoven association fibers Central impulses are for movements not individual muscles

With the foregoing basic generalities in mind the details, some of which modify them, will not be confusing

Innervation in Relation to Speech—To comprehend the numerous and complex factors involved in articulate intelligible speech it is necessary to trace the nerve paths concerned The simplified schemata shown in Figures 334 and

335 will lay a foundation for comprehension of the function of speech

For classification and simplification there may be said to be four word-centers, all unilateral and located on the left side of the brain (Fig 335) in right-handed individuals (1) the receptive auditory word-center (AR) in the left superior temporosphenoidal convolution, for perception of words that are heard, (2) the receptive visual word-center (VR) in the posterior part of the left parietal lobe, for perception of printed words, (3) the written-word motor center (MW) in the second left frontal convolution, (4) the spoken-word motor center (MS) in the left third frontal convolution For elucidation these four centers are described separately and sometimes this is of value clinically also, but functionally these centers are so integrated by association fibers that they are best considered together as a language zone When the word center (which is unilateral) determines a certain word is to be spoken the proper impulses do not go directly to muscles, they are transmitted to the cortical executive centers in the lower part of the precentral gyrus of both sides Thence executive impulses go through the corticobulbar fibers to the nucleus ambiguus in the medulla Here are located the origins of the superior and inferior laryngeal nerves that are bundled along with other nerves to make up the two great vagal trunks But it is not only the laryngeal nerves that are activated by the impulses received from the cortical executive centers There go out, synchronously, impulses distributed to the muscles of the chest, the trachea, and the molds of speech including the tongue, palate, pharynx, and lips All of these must coordinate to produce clear, articulate, intelligible words Respiration must be coordinated with the larynx and molds of speech in speaking and singing, but not in writing words

The language ("word ") center is, so to speak, a blank at birth To produce words it must be educated by seeing them printed (visual receptive center, VR Fig 335), and by hearing them pronounced (auditory receptive center, AR) This is entirely apart from education as to the meaning of words, which is chiefly an intellectual matter, regarded as a function of the cortex of the frontal lobes It is not limited to man, the workhorse, ordinarily, knows the meaning of a dozen words or so, and could be taught more, though he cannot express them

Regardless of any past, present, or future controversy as to the precise location of the receptive and motor centers, there can be no question as to the faculty of speech being developed from impulses received through the eye and the ear, nor as to the location of this speech zone on the left side of the brain (in right-handed persons). Over all presides the obviously present but little understood human intellect.

In the foregoing simplified statement we have used for illustration only the efferent motor pathways. These are duplicated by afferent pathways that carry sensory impulses from the respective peripheries to the centers. The simplest as well as the commonest example of the coordinate impulse traverse of both these pathways in one cycle is the *cough reflex*, referred to on a preceding page as the protective function of the larynx. When accumulated pus in the trachea calls for expulsion, the irritation of its presence activates the afferent nerve endings in the tracheal mucosa to send impulses to the medulla whence coordinated efferent impulses come back to all the muscular systems in the chest and larynx required to produce the *hectic blast* and the *tussive squeeze*.² The chief elements in this hectic cycle are these: (1) automatically a deep breath is taken. (2) The glottis is closed by the laryngeal musculature. (3) The intrathoracic pressure is raised. (4) The glottis is suddenly opened, releasing the compressed air, which rushes out as the hectic blast, expelling pus from the trachea and larger bronchi. (5) The muscular compression of the chest squeezes the accumulated pus in the finer bronchi of all segments of all five lobes of both lungs. (6) A deep breath is taken to refill the lower air passages, at the same time this refilling process, along with the diametric enlargement and elongation of the tracheobronchial tree, leavens the viscid tenacious secretions by permeating them with air bubbles, thus increasing the expulsive efficiency of the next hectic cycle.²

The Human Voice—Normally, voice is sound due to vibrations transmitted to the atmosphere by a vibrating column of air issuing from the mouth. The mechanism of its production and control has been described. The *singing voice* differs from the speaking voice by prolongation of vowel sounds in controlled pitches. Approximately, the human voice has an overall range of four octaves. Men's voices may be said to average in range from bass (E 80 d v to E

320 d v) to tenor (C 128 d v to B 480 d v). Women's voices average from contralto (E 160 d v to E 640) to soprano (C 256 d v to C 1024 d v). Ordinarily an individual's voice has a range of about two octaves but by vocal training the range may be extended to about three octaves, and most of a soprano's professional career, unfortunately, is devoted to extending her range upward. All sopranos must attain, at least, "high C" (C3 = 1024 d v), and an octave above "high C" (C4 = 2048 d v) has been attained. Timbre, or musical quality, is due largely to partials. One may be boro with the anatomic qualities of a good larynx, good resonating cavities, and good molds of voice, but these mean only the possession of the instrument. The making of a great singer requires the same number of years of intensive practice in technique and musicianship that are required to make a great violinist.

Mutation—In the male the increase in size of the larynx at puberty deepens the pitch of the voice and, because of the relative suddenness of the change, control for a time is imperfect. The larger larynx includes longer cords and deeper pitch. The voice may break or it may alternate between a higher and a lower pitch. In girls the growth of the larynx is more gradual and there is much less difference in pitch between the voice of girlhood and womanhood. Mutation is physiologic not pathologic.

CONVERSATIONAL VOICE—In ordinary conversation the range of pitch is not as much as an octave. The average man's conversational voice is in the neighborhood of 128 double vibrations which is C below middle C, the average woman's conversational voice is about 256 double vibrations, which is middle C. The vibration cycles usually increase as intensity is increased and they mount high in shrieking and in falsetto. Character of sound that enables one to recognize a familiar voice is attributed to the partials influenced by the resonating cavities and to the muscular action of movable parts due to nerve-cell habits.

A PLEASING VOICE—It is curious to note that, although everyone is more or less influenced by a pleasing voice, no effort is made in training children to develop pleasing qualities of voice. This should be accomplished by constant example as well as by direct instruction. A harsh voice is a handicap throughout life, a pleasing voice an asset. To be pleasing a conversational voice should not be much above

the average pitches mentioned in the preceding paragraph, and should be moderately low in intensity, clear and distinct but not pedantic. For a low voice to be intelligible it should be rather low in pitch also, and the consonants, especially *s*, *t*, and *th*, should be clearly uttered, ultimate syllables and last words in sentences must not be slighted. These points are of especial importance when talking to old people and persons with impaired hearing, and also in using the telephone and in broadcasting.

VOICE IN YAWNING—There is a yawning center and it takes over when the respiratory center is transitorily in abeyance because of fatigue. There is a respiratory cycle with widely open mouth usually accompanied by paoiculation, which serves to empty the veins by muscular pressure. The accompanying laryngeal sound is a phonatory expression of the subjective sensation of fatigue.

VOICE OF THE AGED—The shrill and tremulous voice of many persons in the eighth decade is due to structural changes in the cords and feebleness of nervous and muscular energy. The senile changes in the cords are fibrosis and hyalinization resulting in a coarser, denser connective tissue, with no increase in the squamous epithelial covering. Though these changes are more or less normal for age, repeated inflammatory attacks through the years are probably contributory factors. Senile changes in epithelium probably supply a favorable soil for growth of cancer of the larynx (*q v*).

WHISPERED VOICE—In loud whispering the fricative sound is produced by air passing through the triangular posterior chink. The stroboscope reveals slight vibration of the cordal edges.

Conclusions—In concluding the subject of the physiology of the larynx it may be stated that survival of a patient after total laryngectomy does not belittle the importance of the nine functions of the larynx any more than survival after gastrectomy implies unimportance of the stomach. Survival in both instances is a tribute to nature's power of adjustment to organic loss.

CHEVALIER JACKSON

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EXAMINATION OF THE LARYNX

Generalities—*Pitfalls in Examination of the Larynx*—It is an almost daily occurrence to encounter patients with a history that they had been under treatment for chronic laryngitis for months when suddenly a growth, or a paralysis, or an ulcer was seen in the larynx for the first time. Other patients come with the statement that they had been told their larynx could not be satisfactorily examined. Most of such often serious shortcomings are due to (1) casual, not thorough examination, (2) lack of proper routine, (3) failure to realize that the larynx of any patient, at any age, whose mouth can be opened, can be examined, or (4) failure to see all parts of the larynx, most often the anterior commissure. Careful, persistent practice in the following details will enable any physician to avoid these pitfalls.

Methods—There are four methods of examination of the larynx, and no laryngeal examination can be said to be complete unless all four are used. The laryngologist may be justified in taking the responsibility of saying two are sufficient for a particular case, these two are (1) examination with the laryngeal mirror, and (2) palpation of the laryngeal cartilages. The other two are (3) by direct laryngoscopy, and (4) by the roentgen ray.

Examination of the Larynx with the Mirror.—Fundamental to success in mirror examination of the larynx is the anatomic position of the patient's trunk, neck, and head. Much of the difficulty, chagrin, and many of the pitfalls consequent upon lack of thoroughness, are primarily due to lack of appreciation of this basic anatomic factor. For some unknown reason almost all patients assume a faulty position in which it is practically impossible to obtain a good view of the larynx with the mirror (Fig. 336).

Step 1 Before asking the patient to open his mouth he should be asked to sit all the way back in his chair. Then the examiner, with a hand on each lateral angle of the patient's lower jaw, gently draws the patient's head forward to the position shown in Figure 337.

Step 2. The patient should then be asked to open his mouth. Without the insertion of any instrument the distance between the tonsils is estimated as a guide for the selection of the size of mirror to be used. A mirror that is small enough to go between the tonsils without touching either of them is least likely to cause gagging. The patient is then asked to rest a moment.

Step 3. The examiner takes a folded piece of dry gauze in his left hand, the mirror held in his right hand is dipped for a moment in hot water, then wiped dry on the gauze and tested for warmth on the back of the examiner's left hand.

the posterior surface of the epiglottis and the laryngeal orifice come into view as a reflected image in the mirror.

Step 6. Examination and sketching of the reflected image of the larynx is next in order.

Seriatim Examination of the Reflected Image of the Larynx.—The most serious pitfall, often dooming the patient, is that of overlooking an abnormality in its early and curable stage. The first element in avoidance of this tragedy is examination by the seriatim method. To ob-

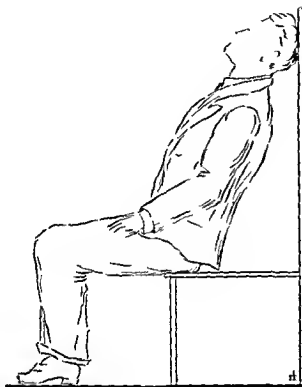


Fig 336

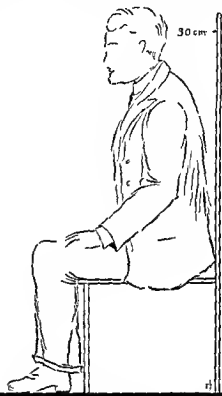


Fig 337

Fig 336 —Pen sketch showing the faulty position in which most patients will place themselves for examination of the larynx. The patient is sitting on the forward edge of the examining chair, the trunk is inclined backward and the head is thrown back as if to have the neck shaved in a barber's chair. It is impossible to get a good mirror view of the larynx with the patient in such a position (see Fig 337).

Fig 337 —Pen sketch showing the proper position of the patient in which he should be placed for examination of the larynx with the mirror. Before he is asked to open his mouth he should be asked to sit all the way back in the chair. His head and shoulders should then be brought forward so that his vertex is about 30 cm, or more, from the vertical which is shown here as the back of the patient's examination chair (see Fig 336).

Step 4. The patient is asked to put out his tongue which is grasped with the gauze held between the thumb and the side of the left second finger over which it is rolled to prevent any painful downward pull against the lower incisor teeth. The tongue should be grasped firmly enough to prevent the patient involuntarily pulling it in. Traction enough is made to draw the back of the tongue away from the pharyngeal backwall (Fig 338). The left index finger holds up the upper lip.

Step 5. The mirror is inserted to a position near the backwall where the back of the mirror will raise the uvula. Touching the back of the tongue or the tonsils will cause gagging. The proper position is reached when

tain the full advantage of this method it should be so firmly established as a habit that it is followed routinely. The routine includes the mental check off of each part as it is examined. Any abnormality noted is recorded. Not more than five minutes are needed if no abnormality is found. If abnormality is present additional time will be necessary for special study and making sketches. The following is a good order of seriatim examination to establish as a habit.

to the cords looks to be 1 or 2 cm. in an adult, whereas it is nearer 6 cm. (4) A serious illusion, and one that is difficult to eradicate from the mind, is the misconception that the vocal cord has an outer border. This is due to the shadow of the overhanging ventricular band (Fig. 339) (5) Because of the angulation of the visual axis

ventricular band, which in all cases, hides the ventricular floor and walls from view in the mirror. In fact no one would even suspect the existence of a ventricle from the reflected image (Fig. 340). Calling attention to these seven illusions should not be mistaken as belittling the value of examination with the

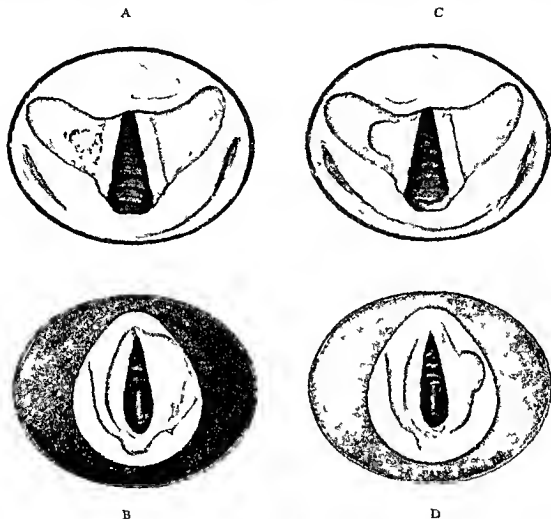


Fig. 340—Drawing from life illustrating the illusion of an external border of the vocal cord. At A is shown the reflected image of the larynx of a man aged sixty three years. There is a sharp dark line of demarcation apparently the outer border of each vocal cord. On the right ventricular band there is a fungating ulcer. At B the right ventricular band with the growth on it (direct view same patient recumbent) is lifted with the tip of the laryngoscope hiding the growth but exposing the floor of the ventricle on which no external border of the vocal cord is visible. It was an illusion created by the shadow of the overhanging ventricular band the shadow line disappeared when the band was lifted. At C the ulcer has been punched out of the overhang again showing the disappearance of the illusory shadow. At D is the direct appearance after excision demonstrating conclusively the fact that there is no external border of the vocal cords (see Fig. 339).

in the mirror any small lesion on a vocal cord seems to be located farther posteriorly than it really is (6) The overhang of the epiglottis in many cases hides the anterior commissure from view. This has cost many a patient his life by hiding from view a cancer in its early and curable stage. (7) The same may be said of the

mirror, it is invaluable and always will be, but it is necessary to call attention to the illusions for the anatomic accuracy so important in diagnosis and treatment. Foreknowledge of these illusions will prevent bewilderment and failure when commencing the use of the direct laryngoscope which gives a

true image of the anatomy, normal or pathologic, as it exists in the particular patient under examination

The various endolaryngeal landmarks are shown in Figure 338. The dark central triangle is the *glottic aperture*. In illustrations, for completeness, the anterior wall of the *conus elasticus* and trachea are shown, but in serial examination, and in the sketch recording it is better to form the habit of ignoring the sub-

mination. Intense illumination blanches the mucosal appearance so that a dark inflammatory mucosa looks normal or paler than normal, even anemic. It is the light that reaches the endolaryngeal surface that determines color perception. Four factors enter into this: (1) tissues cutting down the area of admission of the beam act like diminution of shutter aperture on a camera, (2) source of light, (3) reflecting efficiency of the throat mirror, and (4) focal

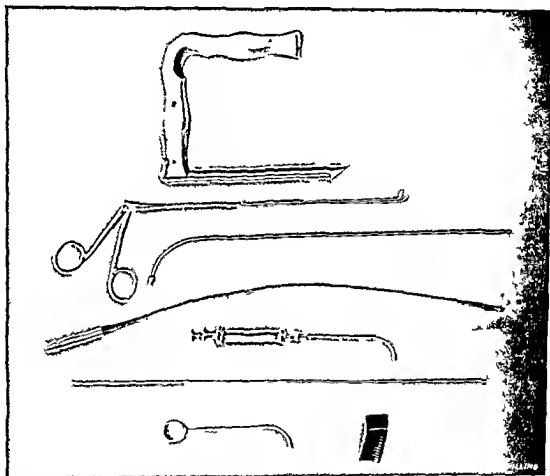


Fig. 341—Instruments for diagnostic laryngoscopy. From above downward: Anterior commissure laryngoscope (C. L. J. model); Tissue forceps; Aspirating tube, metallic; Aspirating tube, silk woven; Laryngeal syringe; Lukens model; Sponge carrier; Mouth opener (C. L. J. model); Bite block.

glottic region for the moment while noting the glottis as a silhouette—the *glottic silhouette*. The less it is illuminated the better for this purpose. This silhouette is an important part of the record for diagnostic purposes in relation to diseases of the larynx (Figs. 542, 543). The cords look thin, sharp edged and whitish, often pearly white in health, in contrast with the delicate velvety pink of the laryngeal mucosa. Color depends much on the degree of illu-

distance of the head mirror. Commercial circuits vary in voltage, distance from the condensing lens as well as from the concave head mirror greatly varies intensity, surfaces of lens and both mirrors must always be clean and free from condensation or imperfection. The surface and the silvering of laryngeal mirrors soon are dulled by repeated sterilization. The examiner should form the habit of preliminarily gauging the intensity of light, if it cannot be

made of usual intensity due allowance must be accordingly made

Examination of Bedfast Patient with the Mirror—In case of a patient who for any reason, such as fracture or extreme degree of illness, must be examined recumbent, the same relative positions of head and trunk apply. The patient should lie flat with the head raised on a pillow, but not extended. In thousands of examinations of the larynx with the mirror in patients with typhoid fever, this position was

mologist knows that oblique illumination enables him to see otherwise invisible lesions on the cornea. For best determination of color the light should always be adjusted to the same intensity. Over illumination blanches the apparent color of the mucosa. All instruments should be sterile and sterile technic should be strictly followed as a routine to prevent carrying specific infections like syphilis and tuberculosis as well as pyogenic infections from one patient to another.

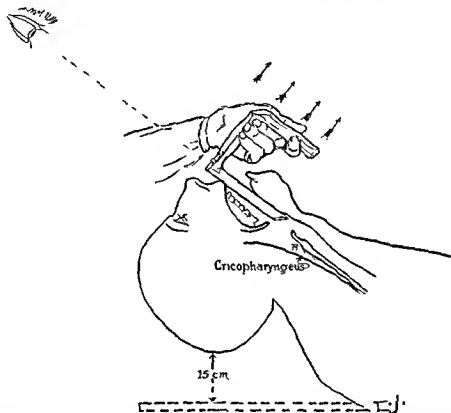


Fig. 342.—Schematic illustration of the fundamental mechanical principles of (1) examining the larynx, (2) illuminating the lingual death zone and (3) lining up the airway. Every detail—the position of every finger, the lifting in direction of darts—all are important. As with any other tool there is one best way to grasp the laryngoscope. It is shown here. This grasp should be practiced until it becomes a habit, and always with the left hand.

found even more satisfactory than the usual sitting position.¹ A self illuminated mirror is convenient.

Direct Laryngoscopy—This procedure is, in principle, a specular examination. It gives an undistorted view of the larynx for diagnosis and yields direct access for diagnostic tests of passive mobility for the taking of specimens of tissue or secretions, and for local medication and operations. The direct laryngoscope (Fig. 341) is the instrument used. It has a distal light which, being at one side, gives the great advantage of oblique illumination. Every ophthal-

Contraindications—Practically there is only one contraindication, namely, disease of the spine. Examination with the mirror and external palpation should always precede direct laryngoscopy.

Preparation of the Patient—The essentials are an empty stomach and a mind free from apprehension. The reason for the empty stomach is that otherwise vomiting would occur when the back of the tongue is touched with an instrument. To quiet apprehension we use what we call the "sermon on relaxation."²

A SERMON ON RELAXATION—If the patient

would relax to limpness, exposure of the larynx would be easily obtained, simply by lifting the head with the lip of the laryngoscope passed below the tip of the epiglottis (as in Fig. 342) and no holding of the head would be necessary. But only rarely is a patient found who can do this. This degree of relaxation is, of course, present in profound general ether anesthesia, which is not to be thought of for direct lar-

ation must be explained to him. He should be told to avoid phonating or clenching his fists. If his hands lie flat, muscular tension at the neck will be less. It should be explained to him that if he contracts the muscles of his forearms rigidly by clenching his fists the muscles of his neck will not relax. The tendency of the patient to heave up his chest and assume a false position simulating the opisthotonos position must



Fig. 343.—Position in which the patient must be held for satisfactory direct laryngoscopy. The following details must be memorized and strictly followed. The head must be held high, vertex about 15 cm. above level of the table. Extension must be slight. The left hand with thumb upward and fingers on occiput supports the head and makes the extension. The left elbow rests on the left knee for support. The right arm does not support head or neck, it passes under the neck so the right index finger can hold the bite block between the patient's teeth. Note that it is the assistant's left foot that is on the foot box. His right foot is on the floor, the right knee under the table as much out of the way as possible. The nurse is holding down the patient's shoulders, they must be kept down on the table.

ingoscopy, except when it is used for the purpose of insertion of intratracheal insufflation anesthetic tubes. The muscular tension exerted by some patients in assuming and holding a faulty position is almost as much of a hindrance to peroral endoscopy as is the faulty position itself. A little talk with the patient beforehand is an important part of our regular procedure. The patient does not know how to relax, even if he knows the definition of the word. Relax-

ation must be overcome by persuasion. This position has all the disadvantages of Rose's position for endoscopy. The patient should be told to lie flat on the table and not to raise his chest or shoulders. He must be gently but positively made to understand (1) that while the procedure is alarming, it is absolutely free from danger, (2) that you know just how it feels, (3) that you will not allow his breath to be shut off completely for too long a time, (4) that he can

help you and himself very much by paying close attention to breathing deeply, quietly and regularly, and (5) that he must not draw himself up rigidly as though "walking on ice", he must be easy and relaxed. This getting of the patient's confidence and ridding his mind of apprehension is one of the most important things in *working without general anesthesia (q v)*

Position of the Patient—Direct laryngoscopy can be done with the patient in the sitting position but we do not use it because the recumbent posture affords better control. Every detail of the illustrations (Figs 342, 343, and 344) should be precisely followed. The writer suggests that the beginner should postpone at-

while giving and taking orders. Every member of the team knows what he is to do, what every one else is to do, and when. Everything goes quietly and smoothly. This does not mean that the looking into the larynx is done hastily. It does mean that no time is wasted in the other details. There is one exception as to time taken to look into the larynx, namely, in case of a child in whom many direct laryngoscopies will be necessary for treatment, in cicatricial stenosis for example. In such cases no attempt whatever is made to look at the larynx until the child learns by experience that direct laryngoscopy is not painful.^{2 3 4} At the first "laryngoscopy" we touch the tongue as with a



Fig. 344—Direct laryngoscopy. The green tint of sterile drapery is of utmost importance.

tempts to improve on this position until he has used it in at least a hundred examinations or direct laryngoscopic operations.

Laryngoscopy without Anesthesia—No anesthetic, general or local, is necessary for children and it is essential that the examiner and his assistants be well trained in doing this procedure without anesthesia because of the cases in which the use of any anesthetic is contraindicated, for example in acute laryngotracheo-bronchitis, diphtheria, and extreme dyspnea. The most important thing in doing direct laryngoscopy without anesthesia in children is teamwork by a well organized team thoroughly trained to work together. Not a second is wasted fumbling around in ineffectual attempts

tongue depressor. At each subsequent "laryngoscopy" we go a little farther until at the fourth or fifth seance we reach and really inspect the larynx. If at any future laryngoscopy it is necessary to operate or otherwise give pain, the child is told "now this will hurt a little bit, but it will be over in a moment," and we see to it that it is over in a moment. The child learns to have confidence in us. We never betray that confidence.

Anesthesia in Children—As stated in the preceding paragraph, no anesthetic, general or local, is necessary for direct laryngoscopy in children. We have the testimony of older children who have had many laryngoscopies for treatment of chronic cicatricial stenosis to the

effect that the application of the anesthetic is annoying and that they would prefer to have us go ahead with the laryngoscopy and be promptly done with it.³ We have their testimony that the procedure does not really hurt." They climb on the table voluntarily. It certainly is no more annoying than digital examination of the nasopharynx for adenoids, and no one uses anesthetic for that.

If, regardless of the above considerations, a local anesthetic is desired in a particular case, the drug to be used must be carefully selected and applied with particular caution. Cocaine is the most efficient anesthetic for topical application, but it is dangerous in children under about six or eight years of age. Above that age it may be used, if proper safeguards and supervision are continuously maintained. Any physician or surgeon applying cocaine as a local anesthetic for any purpose should be on the alert to detect the earliest toxic signs and should have prepared beforehand, ready for instant use, the proper antidotes (see "Toxic Effects", p. 784). The toxic action is so sudden in onset and so rapid in progress that not a second should be lost in combating it. Its chief use is for the first few examinations. In chronic cases in which many treatments are likely to be necessary it is better to use only larocaine or nothing at all locally. Pontocaine, though not so efficient as when used in the conjunctival sac, gives a rather satisfactory local anesthesia for endoscopy in most cases but it is objectionable because it often leaves a sensation of irritation lasting a few days, and, in some patients, it precipitates or causes asthmatic or asthmatic attacks.³ Larocaine is not toxic and is the only safe and satisfactory local anesthetic to use in children. A 10 per cent solution may be sprayed into the pharynx and larynx, a downward pointing tip being used on the atomizer when spraying the larynx. A second application is required after an interval of about two minutes, maximum anesthetic action takes place about two minutes after that.

Anesthesia in Adults—Local anesthetic is used in practically all cases for direct laryngoscopy in adults. The technic we have used for years is as follows:

t. With the patient sitting on the examining chair, in the position shown in Figure 337 cocaine hydrochloride in 10 per cent aqueous solution is sprayed for a second or two into the fauces and pharynx, the spray is directed toward the base of the right faucial pillar, the tongue

being well depressed to expose the right side of the laryngopharynx.

2. After about two minutes the warmed laryngeal mirror is placed so as to expose the larynx to view and, with a sterile Lukens syringe 3 or 4 drops of a 10 per cent solution of cocaine hydrochloride are dropped into the larynx under guidance of the eye.

3. After an interval of about three minutes 3 or 4 additional drops of the same solution are dropped into the larynx.³

The patient is then placed recumbent on the examining table and the larynx is examined with the direct laryngoscope.

The chief features of this technic are accuracy of placement and fractionation.³ It produces the maximum of local anesthesia with the minimum of cocaine, probably not more than 12 or 14 drops of a 10 per cent solution are used for the entire procedure. To get the effect with this small quantity it is essential that every drop from the syringe be seen to enter the larynx. None is wasted by scattering or swallowing.

General Anesthesia—General anesthesia is very rarely justified in children, and not often in adults.³ The various kinds of inhalation anesthetics are inconvenient and many of them dangerous because they are explosive. Avertin (by rectum) is unsuitable because it increases reflexes and causes spastic muscular contractions, besides, its effect is too prolonged. Sodium pentothal given intravenously has been found satisfactory but for safety it requires an anesthetist with technical skill and experience in its use. For the technic of its use see the section on "Anesthesia" (p. 783).

Premedication—If the operator deems it proper to use premedication, and if he keeps constantly in mind the baneful effect of inhibition of the cough reflex, he may be guided by the following considerations. It is of great advantage, in work without anesthesia in children, to give a sedative. In older children and in adults, when cocaine is employed, the amount of this toxic drug can be lessened by the use of a preliminary sedative. The most efficient drug for this purpose is morphine with its usual complement of atropine. The timing is of utmost importance. If given shortly before the laryngoscopy the stimulant effect makes the patient more sensitive and excitable. It should be given long enough beforehand for relaxation and drowsiness to develop. Just how long this may require is somewhat variable. Two hours is usually not too long though an

hour may be enough for some individuals. If the patient shows no sign of drowsiness at the end of an hour an additional half dose without atropine should be given. The amount of the initial dose is variable and dependent on the personal equation of the patient to a greater extent than is generally realized. The resistant combative argumentative type of individual, child or adult, usually requires twice the dose needed for a person of the gentle confiding cooperative type. An average adult, free from organic disease, may be given $\frac{1}{4}$ grain (0.015 gm) of morphine sulfate with $\frac{1}{150}$ grain (0.0004 gm) of atropine. For an adult or a robust, thick necked individual of 170 pounds weight twice this amount of morphine may be given, but the dose of atropine is not also increased. In case of a frail, aged, or very ill adult $\frac{1}{8}$ grain (0.0075 gm) of morphine with $\frac{1}{150}$ grain (0.0004 gm) of atropine sulfate may be sufficient. For children of two years and over it is safe to use young's rule (add 12 to age, divide by the age, result is denominator of fraction whose numerator is 1, representing the required fraction of the adult dose). In any case, adult or child, it is best to give half the dose two hours before laryngoscopy, and to give the other half an hour later. In case of any idiosyncrasy the second half dose can be withheld. In some cases, especially when laryngeal operation or bronchoscopy is to be done, the total absence of any effect from the first half-dose may be deemed justification for making the second portion of the dose more than the half of the predetermined dose. Nembutal or one of the other harbiturates may be used, either alone or preceding the morphine, and these drugs have the advantage, in addition to their sedative effect, that they counteract the toxic effects of cocaine. In children under two years of age a few drops of paregoric may be used. The newborn require neither sedative nor anesthetic.^{3 4}

In conclusion it may be said that the personal equation of the examiner, the smoothness of the teamwork, and the elimination of false starts and time wasting enable perfectly satisfactory laryngoscopy in children without any anesthetic, general or local. In adults, the addition of the 'sermon on relaxation,' to the above mentioned factors, minimizes the amount of local anesthetic that need be used. Victims of impending asphyxia need nothing but prompt and skilful instrumentation.

Instrumentation—There are two steps in laryngoscopy namely, (1) finding the epiglottis, and (2) exposure of the laryngeal interior to view. It is best to have a conception of these two steps distinctly and always in mind, in order to develop a proper technic. After the larynx is exposed the glottis must be promptly visualized, because, in emergencies, locating it promptly may save life. Having located and visualized the glottic silhouette, the operator proceeds to examine the laryngeal structures *seriatim* in the same order as given for examination with the mirror.

The following description of the two steps presents the standard technic.²

First Step The spatular end of the laryngoscope is introduced in the right side of the patient's mouth, along the right side of the anterior two-thirds of the tongue. It is a faulty method to introduce the laryngoscope over the dorsum of the tongue, since in order thus to elevate this resistant and powerful muscular organ considerable force is required. Such an exercise of force may be entirely avoided by crowding the tongue over to the left. When the posterior third of the tongue is reached, the tip of the laryngoscope is directed toward the midline, and the dorsum of the tongue is elevated by a lifting motion imparted to the laryngoscope. The epiglottis will then be seen to project down into the top of the endoscopic field.

Second Step The spatular end of the laryngoscope should now be tipped backward toward the posterior wall of the pharynx passed posterior to the epiglottis, and advanced about 1 cm. The larynx is now exposed by a motion that is best described as a suspension of the head and all the structures attached to the hyoid bone on the tip of the spatular end of the laryngoscope (Fig 342). Particular care must be taken at this stage not to pry with the upper teeth as a fulcrum, but rather to impart a lifting motion with the tip of the speculum without depressing the proximal tubular orifice. It is to be emphasized that, while some pressure is necessary in the lifting motion, great force should not be used; exposure of the larynx is more a feat of skill and knack than of great strength. The first view is likely to find the larynx in a state of spasmodic closure and affords an excellent demonstration of the fact that the larynx can be completely closed without the aid of the epiglottis. Usually little more is seen than the two rounded arytenoid masses and, anterior to them, the ventricular bands in more or less close apposition hiding the cords. With deep general anesthesia or thorough local anesthesia the spasm may not be present. It is better not to ask the patient to take a deep breath, nor to request a phonatory effort, very soon the larynx will open widely and the cords be revealed. If the anterior commissure of the larynx is not readily seen, the lifting motion and elevation of the head should be increased and if there is still difficulty in exposing the anterior commissure the assistant holding the shoulders should with the index finger externally on the neck depress the thyroid cartilage. The interior of each ventricle should next be inspected by lifting each ventricular band in turn (Fig

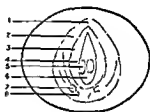


Fig 345—Upper, Schematic presentation of the normal larynx as seen in the laryngeal mirror during quiet breathing. It is composite in the sense that it is made up of more regions of the larynx than can usually be taken in at one time and at one position of the laryngeal mirror, and, moreover, there are wide variations within normal limits, in fact, no two larynges are exactly alike. It is not exactly symmetrical, neither is the normal larynx. It is a misleading image in that (a) it gives no impression of real depth, (b) nor of a ventricle, (c) the cords seem thin and sharp at their edges, (d) the cords seem white whereas really they are pink, (e) the image is reversed anteroposteriorly but laterally it is unreversed. As is often the case the anterior commissure is hidden by an overhanging epiglottis. 1 Glosso-epiglottic ligament, median 2 Epiglottis, anterior surface 3 Ventricular band. Hidden beneath it is the ventricle, of which it forms the roof 4 Vocal cord, right 5 Pyliform sinus 6 Aryepiglottic fold 7 Cuneiform tubercle usually containing the cartilage of Wrisberg 8 Glottis 9 Cartilage of Santorini capping the apex of the arytenoid 10 Posterior commissure.

Lower, Schematic presentation of the same larynx as seen brilliantly illuminated through the laryngoscope. The image is quite different. The epiglottis is bled out of sight, the anterior commissure is exposed to view, the normally thick edges of the cords are seen, the cords are pink, one gets an impression of a ventricle or at least an overhang of the ventricular bands, the aryepiglottic folds are pressed apart as the laryngeal orifice is held open by the tube mouth. Because the distal light is right at the field there are no shadows cast as is the case with light reflected down from a mirror high above. 1 Anterior commissure 2 Ventricular band 3 Floor of ventricle 4 Vocal cord 5 Left bronchial orifice (often only the right orifice is in line of vision) 6 Posterior tracheal wall 7 Cuneiform tubercle 8 Arytenoid prominence 9

340) The anterior-commissure laryngoscope should be passed through the glottis for the examination of the subglottic area, usually the trachea is inspected also. The lower border of a growth if any, should always be determined. If there is much obstruction to breathing the patient will appreciate the passing of the laryngoscope through the glottis to admit air.

After the extrinsic, intrinsic, and subglottic areas of the larynx are examined, the laryngoscope is passed into the hypopharynx (H, Fig. 342) for the exploration of the "party wall"—that is, the posterior wall of the larynx which constitutes the anterior wall of the hypopharynx. Edema of the arytenoids seen in the mirror is often caused by a cancerous lesion in the hypopharynx, but whether or not there is such reason for suspicion the hypopharynx should always be examined (see Hypopharyngoscopy").

Difficulties of Direct Laryngoscopy—If the standard technic just described be correctly followed there should be no difficulty after the

spatula being directed toward the midline when the posterior third of the tongue is reached. The epiglottis must always be identified before any attempt is made to expose the larynx.

5 When first inserting the laryngoscope to find the epiglottis, great care should be taken not to insert too deeply lest the epiglottis be overridden and thus hidden.

6 After identification of the epiglottis, too deep insertion of the laryngoscope must be carefully avoided lest the spatula be inserted back of the arytenoids into the hypopharynx.

7 Exposure of the larynx is accomplished by lifting forward the epiglottis and the tissues attached to the hyoid bone (Fig. 342) and not by prying these tissues forward with the upper teeth as a fulcrum.

8 Care must be taken to avoid mistaking the aryepiglottic fold for the epiglottis itself. This is most likely to occur as the result of rotation of the patient's head.

9 If the laryngoscope is too long in place, accumulated saliva will trickle down the trachea and cause

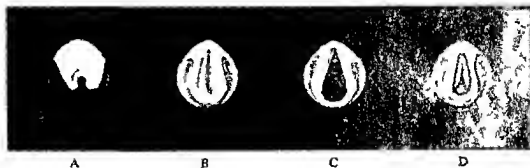


Fig. 345a—Brush sketches from life illustrative of the laryngoscopic appearances in a child when examined without anesthesia, general or local. The first stage (A) is presentation of the epiglottis. The form of epiglottis varies, but this omega (Ω) shape is common. Lifting with the laryngoscope, in the direction of the darts shown in Figure 342, brings into view the closed laryngeal orifice (B). A moment later the child takes a deep breath and the larynx opens widely, as sketched at C. The three foregoing views are normal. At D is shown the swollen appearance in acute laryngotracheobronchitis. The subglottic airway is narrowed by masses of swollen tissue, one under each cord.

knack is acquired. Education of the eye and the fingers is essential. As with any medical or surgical procedure, abundant experience with the normal appearance is essential for recognition of the abnormal. If difficulties are encountered the examiner should ask himself which one of the following axioms he has disregarded:

Rules for Direct Laryngoscopy 2

1 The laryngoscope must always be held in the left hand, never in the right.

2 The operator's right index finger should be used to retract the patient's upper lip so that there is no danger of pinching the lip between the instrument and the teeth.

3 The patient's head must always be exactly in the middle line, not rotated to the right or left, nor bent over sideways, and the entire head must be forward with extension at the occipito-atloid joint only.

4 The laryngoscope is inserted to the right side of the anterior two-thirds of the tongue, the tip of the

cough. Swallowing is almost impossible while the laryngoscope is in position. The secretions should be removed with the aspirator.

Normal Direct Laryngoscopic Appearances in Adults—When the larynx was examined with the direct laryngoscope by the older group of laryngologists, who had never seen the larynx except with the mirror, the direct image was so different that for a long time direct laryngoscopy was neglected. The illusions cited under "Interpretation of the Reflected Image" had implanted such a false conception of the larynx that the true appearances were deemed false (Figs. 340 and 345). The cords are not pearly white and they are longer than they seemed, the ventricular bands are not lumps, they are more like folds, the epiglottis is absent from the foreground of the picture, and its contrast is missing, the distance from the tip of

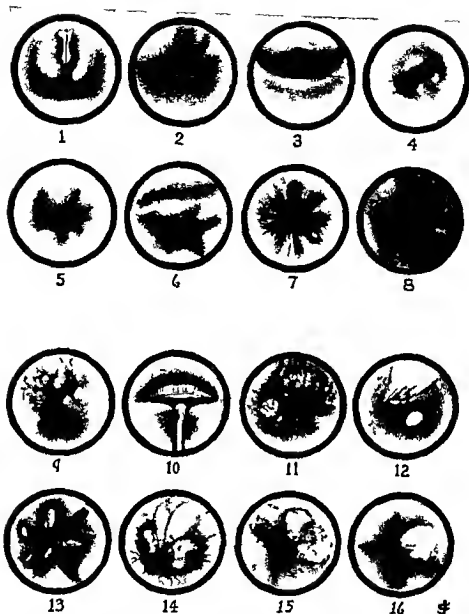


Fig. 346—Esophagoscopic views from color drawings from life. 1 Direct view of the larynx and laryngopharynx in the dorsally recumbent patient the epiglottis and hyoid bone being lifted with the direct laryngoscope. The spasmodically adducted vocal cords are partially hidden by the overhang of the spasmodically prominent ventricular bands. Posterior to this the aryepiglottic folds ending posteriorly in the arytenoid eminences are seen in apposition. The esophagoscope should be passed to the right of the median line into the right pyriform sinus represented here by the right arm of the dark crescent. 2 The right pyriform sinus in the dorsally recumbent patient the eminence at the upper left border corresponds to the edge of the cricoid cartilage. 3 The cricopharyngeal crescent at the orifice of the esophagus in the dorsally recumbent patient the cricoid cartilage being lifted forward with the laryngoscope. The lower (posterior) half of the lumen is closed by the fold corresponding to the orbicular fibers of the cricopharynx which advances continuously from the posterior wall except when relaxed during swallowing. (Compare 10). This view is not obtained with an esophagoscope. 4 Passing through the right pyriform sinus with the esophagoscope dorsally recumbent patient. The walls seem in tight apposition, and at the edges of the slitlike lumen, bulge toward the observer. The direction of the axis of the slit varies depending on the position of the folds. 5 Cervical esophagus. The lumen is not patent during inspiration as it is lower down and it closes completely during expiration. 6 Thoracic esophagus dorsally recumbent patient. The ridge crossing above the lumen corresponds to the left bronchus. It is seldom so prominent as in this patient, but can always be found if searched for. 7 The normal esophagus at the hiatus. This hiatus pinching is often mistaken for the cardia by esophagoscopists. 8 View in the stomach with the open tube gastroscope. The form of the folds varies continually. 9 Sarcoma of the posterior wall of the upper third of the esophagus in a woman of thirty-one years. Seen through the

the epiglottis to the glottis is seen as the true depth.

Hypopharyngoscopy—At every diagnostic direct laryngoscopy the hypopharynx should be examined. By inspection of the "party wall" between the hypopharynx and the larynx the laryngeal examination is completed. This is essential in every case of slightest abnormality referable to the larynx or to the swallowing function, to omit it is a serious matter in cases of present or suspected malignant disease. Not to record the shape and action of the *cricopharyngeal crescent* (3, Fig. 346) means ignoring an important part of the diagnostic study in cases of laryngeal paralysis and related syndromes (q v). When little lifting effort is used the laryngoscope will not show the crescent, the hypopharynx will be found closed from the cervical esophagus by the tonic contraction of the *cricopharyngeus* (H, Fig. 342).

Normal Direct Laryngeal Appearances in Children—The appearances are quite different in children, as compared to those of adults. The epiglottis is flexible and elusive, the whole interior of the larynx is invisible until a deep breath is taken (A, B, C, D, Fig. 345a).

Inducing a Child to Open His Mouth—If a child clenches his teeth the mouth opener (Fig. 341) should be insinuated between the upper jaw and the mandible back of the teeth and passed backward toward the pharynx. As soon as the instrument touches the back of the tongue retching will cause the mouth to open.

Direct Laryngoscopy in Chronic Diseases of Childhood—When the prospects are that many future direct laryngoscopies will be necessary great care should be taken not to frighten the child (see "Laryngoscopy without Anesthesia").

Direct Laryngoscopy in the Newborn—The technic of inspection of the larynx and of aspiration of secretions from the pharynx, larynx,

and trachea is much simpler in the newborn than in older children or adults. All the structures are flexible and there is little muscular resistance. The procedure is harmless provided the delicacy of infant tissues is borne in mind and provided due attention has been given to acquiring proper technic. The child is brought crosswise to the edge of the bed. The head is held elevated to proper position (Fig. 343) by an assistant who uses his right index finger as a bite block. The laryngoscope is inserted as described on a previous page and with it the epiglottis and base of the tongue are gently lifted in the direction shown in Figure 342 exposing the larynx to view. Secretions are always present and should be aspirated with the aspirating tube to give a clear view, as well as to clear the airway (Figs. 358, 469). Gentleness is fundamental and familiarity with the technic and appearances are best acquired on the newborn cadaver, which is available in anatomic laboratories. This simple technic should be taught to the undergraduates in all medical schools. This would go a long way in still further reducing infant mortality at birth, in which department of medical science so much has been done in recent years (see "Asphyxia Neonatorum").

External Palpation of the Larynx—It is customary to palpate the neck, but palpation of the laryngeal cartilages is not taught as it should be. All of the cartilages of the laryngeal framework can be palpated, just as the orthopedic surgeon palpates each of the eight bones of the carpus. The digital palpation of the laryngeal cartilages should be done for (1) the detection of abnormalities in texture and form, (2) to test mobility as bearing on cricothyroid arthritic fixation, denoted by absence of rocking movement, and (3) for the elicitation and accurate localization of tenderness, as bearing on the diagnosis of such pathologic processes as

esophageal speculum, patient sitting. The lumen of the mouth of the esophagus, much encroached upon by the sarcomatous infiltration, is seen at the lower part of the circle. 10. Coin (half-dollar) wedged in the upper third of the esophagus of a boy aged fourteen years. Seen through the esophageal speculum, recumbent patient. Forceps are retracting the cricopharyngeal crescent preparatory to removal of the coin. 11, Fungating squamous-celled epithelioma in a man of seventy-four years. Fungations are not always present, and are often pale and edematous. 12, Cicatricial stenosis of the esophagus due to the swallowing of lye in a boy of four years. Below the upper stricture is seen a second stricture. An ulcer surrounded by an inflammatory areola and the granulation tissue together illustrate the etiology of cicatricial tissue. The fan-shaped scar is really almost linear, but it is viewed in perspective. Patient was cured by esophagoscopy dilatation. 13, Angioma of the esophagus in a man of forty years. 14, Syphilitic ulcer of the esophagus 26 cm. from the upper teeth in a woman of thirty-eight years. Two scars from healed ulcerations are seen in perspective on the anterior wall. Branching vessels are seen in the livid areola of the ulcers. 15, Tuberculosis of the esophagus in a man of thirty-four years. 16, Leukoplakia of the esophagus near the hiatus in a man aged fifty-six years.

perichondritis necrosis arthritis and fixation (qv) For this the fingers must be trained by this training is a deplorable shortcoming Training is obtainable by making palpation a part of

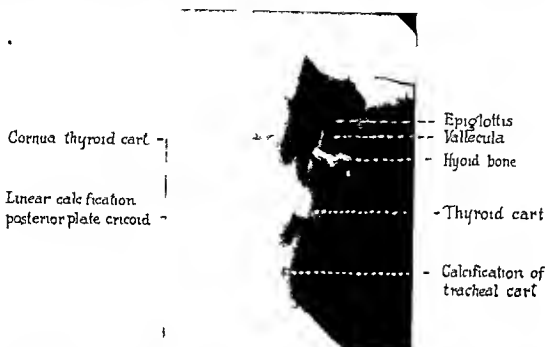


Fig 347—Lateral roentgenogram showing how laryngeal cartilages may be identified by calcified areas as landmarks. One such area in the posterior plate of the cricoid might easily be mistaken for a foreign body especially a bone lodged in the hypopharynx (Roentgenogram by W. Edward Chamberlain and Barton R. Young)

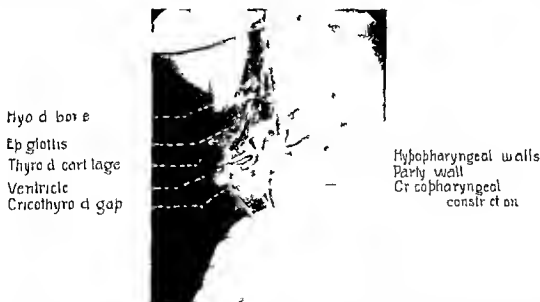


Fig 348—Delineation of the anterior mucosa covered walls of the larynx and the relation to the laryngeal cartilages made by instilling a few cubic centimeters of lipiodol with a laryngeal syringe. This is a standard normal delineation useful for comparison with similarly made films for diagnosis and planning of treatment. Tumors, fibrosis, ulcers, ulcerogranulomas, and perichondral lesions are clearly and accurately outlined by this method.

the palpation of hundreds of normal living larynges after having palpated and studied larynges of many anatomic specimens. Lack of the examination of every patient, some of whom it might be added, will certainly benefit from its revelations.

Röntgen-Ray Examination of the Larynx.—By *fluoroscopy*, in adults the arytenoid cartilages are sufficiently radiopaque to be seen in movement. *Röntgenograms* in the lateral projection will show irregular calcification of the thyroid and cricoid cartilages, from which the outline of these cartilages can be determined (Fig 347). For proper interpretation a large comparative series of such roentgenograms is necessary because of individual variations in location and extent of radiopaque deposits. *Delineation* of the laryngeal lumen (Fig 348) is obtained by dropping lipiodol into the larynx after, and in the same way as, cocaine anesthetic solution (p 436). (See "Anesthesia in Adults.") *Plano-*grams are quite useful, by special apparatus and

regular, habitual order. The foregoing refers to otolaryngologic regional examination. The subject is additionally considered under "General Phases of Examination and Treatment of Patients."

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- 3 Jackson, Chevalier, and McReynolds, George S. *Anesthesia for Otolaryngology*, Ann Otol, Rhin & Laryng, 49 1048, 1940

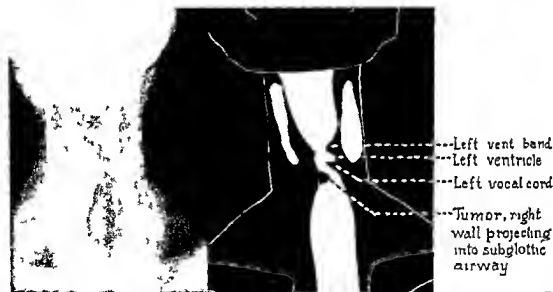


Fig 349—Planigram showing left side of larynx normal in contrast to a neoplastic infiltration of the right side. Biopsy showed the tumor to be a carcinoma. (Planigram by W Edward Chamberlain and Barton R Young)

technic the shadow of the spine is eliminated (Fig 349).

Complete Examination of Every Patient.—In the foregoing pages we have detailed the examination of the larynx, and preceding those pages there are detailed the methods of examination of related regions. It should be emphatically stated that no one of these regions should be examined without also examining all the others. In every case the examination should include the mouth, teeth, tongue, tonsils, palate, pharynx, nasopharynx, external nose, nasal chambers, sinuses, auricle, auditory canal, and drum membrane. As a concession to the patient's satisfaction the start may be made in the region of the chief complaint, but the examination of the other regions should follow in a

- 4 Jackson, Chevalier. *Peroral Endoscopy and Laryngeal Surgery*, The Laryngoscope Company, St Louis, 1915
- 5 Jackson, Chevalier, and Jackson, Chevalier L. *Diseases and Injuries of the Larynx*, ed 2, New York, The Macmillan Company, 1942
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HOARSENESS

Hoarseness is, obviously, a symptom and not a disease. It is important, however, that it be considered separately to save much repetition and to emphasize the importance of prompt in-

vestigation because it is frequently a precursor of serious disease in the larynx or elsewhere

Definition—Hoarseness is a quality of voice that is rough grating harsh more or less discordant and lower in pitch than normal for the individual. Like clarity of voice it is a purely relative term. *Huskeness* is properly limited to superlative degrees of hoarseness of dry quality. *Raucous* is literal Latin for hoarse but commonly implies loudness also and is applicable to naturally coarse crude voices.

Pathology—Setting the air column in vibration by the larynx is a purely mechanical process. Anything that impairs the perfect working of this mechanism produces hoarse-ness.

Mechanical elements are: (1) approximation of the cords; (2) firming or hardening of the cordal edges which is a physiologic change in resiliency; and (3) vibration of the cords. Approximation may be impaired by (a) tumor secretion, or other substance between any part of the two cords; (b) fixation or limitation of range of movement of the cricoarytenoid joint; (c) paralysis or impaired power of movement in the laryngeal musculature; (d) cicatricial concavity of the edge of either vocal cord. Firming may be interfered with by (a) thickening or other tissue change in the cordal edge; (b) concavity of this edge; (c) feebleness of the firming muscles; (d) excessive usurpative approximation of the ventricular bands. Vibration may be impaired by (a) inflammatory neoplastic or other tissue change in the cord; (b) secretion or pedunculated tumor acting as a damper; (c) feebleness of the hardening muscles; (d) in complete approximation; (e) usurpative prior approximation of the ventricular bands.

The pathologic processes participating in these mechanical impairments are elsewhere herein described in connection with the various forms of laryngeal disease in which hoarseness is a symptom.

Differential Diagnosis of Hoarseness—It is a deplorable fact that the laity make a diagnosis of chronic laryngitis in any case of chronic hoarseness and unfortunately the physician may drop into the pitfall of accepting this diagnosis. In reality the diagnosis of laryngitis should never be made until more serious conditions have been excluded. In case of a patient with the symptoms of hoarseness the physician is confronted with a large problem on the early and correct solution of which the patient's life may depend. The following general list of diseases associated with hoarseness was compiled from the records of our clinic up to and including the year 1928. Since its publication in 1929 it has been in constant and satisfactory clinical

use by ourselves and our pupils as a categorical checking list of diagnostic possibilities.

- | | | |
|----|---|---|
| 1 | Malignant neoplasm | } The lesion in these conditions may be located in the larynx, in the trachea, in the bronchus, in the central nervous system |
| 2 | Benign neoplasm | |
| 3 | Tuberculosis | |
| 4 | Lupus | |
| 5 | Syphilis | |
| 6 | Hemorrhage | |
| 7 | Recurrent paralysis | |
| 8 | Angioneurotic edema | |
| 9 | Leprosy | |
| 10 | Scleroma | |
| 11 | Gout—mediastinal cervical or endotracheal | |
| 12 | Aneurysm | |
| 13 | Thickened pleura | |
| 14 | Tabes | |
| 15 | Disseminated sclerosis | |
| 16 | Glossolabio-pharyngeal paralysis | |
| 17 | Syngomyelia | |
| 18 | Syndrome paralytic | |
| 19 | Nephritis | |
| 20 | Protrusion of the ventricle | |
| 21 | Pachydermia | |
| 22 | Dislocation (a) thyrohyoid (b) crico-arytenoid (c) cricothyroid | |
| 23 | Foreign body | |
| 24 | Laryngospasm, laryngomyopathy feeble tendons | |
| 25 | Vocal nodules | |
| 26 | Perichondritis | |
| 27 | Crico-arytenoid arthritis | |
| 28 | Crico-arytenoid ankylosis | |
| 29 | Cicatrix | |
| 30 | Stenosis (a) congenital (b) acquired | |
| 31 | Pericardial effusion | |
| 32 | Dilatation of left auricle | |
| 33 | Trauma anatomic | |
| 34 | Phonation with ventricular bands or other factors | |
| 35 | Papilloma | |
| 36 | Diphtheria—local toxic glandular pressure | |
| 37 | Puberty | |
| 38 | Laryngismus stridulus | |
| 39 | Screamer's nodes | |
| 40 | Vocal abuse | |
| 41 | Feebleness of laryngeal musculature | |
| 42 | Influenza | |
| 43 | Acute laryngitis | |
| 44 | Chronic laryngitis—infiltrative atrophic, subglottic etc | |
| 45 | Glanders | |
| 46 | Mycosis | |
| 47 | Anthrax | |
| 48 | Acromegaly | |
| 49 | Hysteria | |
| 50 | Trauma psychic | |
| 51 | Myasthenia laryngealis | |
| 52 | Myositis | |
| 53 | Trichiniasis | |
| 54 | Contact ulcer | |

Differential diagnosis as among these various possibilities in a case of hoarseness requires first a careful record of chief and lesser complaints, family history, previous medical history.

tory, and present illness, next an examination of the larynx by all four methods as given on a previous page, and finally a general medical examination and a serologic test for syphilis, bacteriologic studies, and, in some cases, biopsy (See under "General Phases of Examination and Treatment of Patients") Other phases of the diagnostic problem of hoarseness will be found in connection with the consideration of particular diseases

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ACUTE INFECTIOUS LARYNGITIS

Acute infectious laryngitis may be defined as an acute inflammation of the laryngeal mucosa due to infection. Synonyms are *angina laryngea*, *acute laryngitis*, *cold in the throat*. Simple laryngitis is a much used but inappropriate term. As part of the common cold, acute infectious laryngitis is one of the most frequent of all human diseases. Unassociated with acute rhinitis it is much less common. Probably no human being, in temperate zones, attains adult age without having had attacks of this disease of the larynx.

Etiology—That the disease is an infection is indicated by its contagiousness, epidemic character, symptomatology, definite course, self-limited termination, and by the immunity it confers. When we come to determination of the infective agent we are confronted with a problem that has been subjected to prolonged and thorough investigation by many of the most capable research workers since the dawn of scientific medicine without conclusive or perfectly satisfactory results. Most investigators now agree that the common cold, of which acute infectious laryngitis usually forms a part, is due primarily to a filtrable virus often in symbiosis with one or more of the common pathogenic organisms. The latter

the most often found are *Haemophilus influenzae*, *pneumococci*, *hemophilus*, *staphylococci*, and *hemolytic staphylococci*, but other bacteria also are found. There seems little doubt that there is secondary invasion often by energization of resident bacteria already present. Probably particular strains of bacteria are as important as particular kinds, both the primary and the secondary causative agents. Bacteriology at some future day will have more definite data bearing on this aspect. Of the predisposing causes, the most common next to frequent chronic disease of the nasal accessory sinuses and, especially in children, of the lymphoid tissue in the fauces, pharynx, and nasopharynx. In these regions the bacteria above mentioned are probably harbored during the interval of temporary immunity conferred by an acute attack, ready to become active as soon as the immunity wanes. The infected secretions of the pharynx, every night, find their way into the larynx of every person who sleeps on his back. Mouth breathing is undoubtedly a contributing cause of all inflammatory diseases of the larynx. There are individual variations in susceptibility that seem to bear no relation to any known causative factor. Some women are rarely affected, few men have this relative immunity. The disease is much more frequent in the winter season, which probably explains the name of "cold." It will take many years and a new generation to eradicate the idea that cold air and drafts (air in motion) are exciting causes. The same may be said of damp air, notwithstanding the fact that a high percentage of relative humidity is beneficial to mucosal surfaces, whereas the dry air of our overheated rooms in winter is probably a predisposing factor in inflammatory conditions of the mucosa. Climate has an influence even apart from humidity and temperature. For example, the streptococcal laryngitis so common in the North Temperate Zone is unknown in some tropical countries where both temperature and humidity are high. This might be due simply to localized natural production of bacteriostatic substances.

Pathology—The capillaries are engorged, the tissues are invaded by leukocytes, round-cell infiltration pervades the mucosa and submucosa, transudation of lymph and serum results in swelling, sometimes in excessive edema. Epithelium is exfoliated and joins with the inflam-

matory products to produce a sputum that is scanty at first, compared to the nasal flow, later it increases in amount and toward the end is slightly purulent. As sputum, the secretions are usually mixed with those of the tracheal mucosa. If the acute laryngitis subsides within two weeks, as it usually does, histopathologic changes disappear. If prolonged, a round celled infiltration may become fibrous and remain. Myositis is often as much a cause of hoarseness as is cordal inflammation. Arthritis of the crico-arytenoid joint occurs, though only in the extremely severe cases.

Laryngeal Appearances—The mucosa is red, thickened, and velvety. The cords may be paler than other regions, but often they are as deep a red as the surrounding mucosa. The epithelium soon becomes roughened and eroded. If there is much cough there may be spots of ecchymosis, crimson in color when they appear but they darken gradually. Secretions are scanty at first, they increase slightly and become thicker, but are never as abundant as the nasal secretions in acute coryza. Motility may be impaired by myositis or arthritis.

Symptoms—The chief symptoms are hoarseness, paroxysmal cough, and aching pain in the larynx. The hoarseness may break to aphonia when speaking is attempted. A husky whisper is never lost so long as air is coming through the larynx. If the tracheal mucosa is involved the cough will be strangling in its paroxysms. Fever, up to about 100° F (38.4° C), is usually present the first day or two. If empirin be given the temperature will come down with a profuse sweat. It usually rises again. Slight chilliness accompanies each rise.

Diagnosis—The appearances both reflective and direct are unmistakable to anyone familiar with the appearance of inflamed mucosa. The accompanying acute rhinitis is significant. Diphtheria (*q v*) must always be excluded, as well as the exanthemas.

Complications and Associated Diseases—The most common complication is edema occluding the laryngeal lumen and necessitating tracheotomy to prevent asphyxia. The cause in some cases is excessive virulence of the infections—primary or secondary, or both. In other cases excessive use of the voice during the attack intensifies the inflammation. Nephritis is sometimes an underlying factor. Complications, especially pulmonary, are favored by opiates (they suspend the cough reflex which is the

natural means of pulmonary drainage) and by atropine which synergizes with opiates in increasing viscosity of secretions, rendering them difficult to expel.^{1 2} Tracheitis is a common complication. Rhinitis, pharyngitis, and nasopharyngitis occur as an extension by continuity or as a coincident infection.

Prophylaxis—In theory, avoidance of infection is the obvious prophylactic means, but, practically, its efficient application is extremely difficult. Exposure to droplet infection by a person in the highly infective initial stages of acute infective laryngitis is almost inevitable during an epidemic. Wearing gloves might lessen contactual risks. Immunity from having recently recovered from an attack is the best protection. Next to this is increased resistance constantly maintained by the intake of an excess of all vitamins. As there is no storage of vitamins it is necessary never to miss a daily dose. Green raw vegetables and fresh raw fruits at every meal, every day, are a proven prophylactic measure in most persons. Tomatoes, lettuce, and citrus fruits, especially limes, should be in the dietary every day. These vitamin containing foods will not arrest an attack after it has started, but they are helpful. Fresh air and sunshine are helpful prophylactically, as well as therapeutically. Undoubtedly resistance is increased by a previous summer's sunburn and hardening of the general dermal surface. Frequent bathing at moderate temperatures and spraying the head and back of the neck with cold water from a bath hose every morning also increases resistance. Apropos of prophylaxis it seems proper to mention here that an attack of acute infectious laryngitis should always be regarded as potentially a cause of chronic laryngitis (*q v*) especially if the acute condition is neglected. The patient should be warned of this to obtain his cooperation in treatment and especially in "a daily dose of silence" as we have called it. The widely advertised vaccines for prevention of acute infections of the nose and larynx, commonly called a cold, are of doubtful prophylactic value and those so far offered for oral administration are absolutely devoid of any preventive effect.

Treatment—Fundamental to all treatment is free elimination by bowels, skin, and kidneys. After a hot soapy bath in a warm bathroom the patient should be put to bed in a well ventilated room constantly maintained at a temperature of 72° F (22° C) and at a humidity percentage of

about 50 A compound cathartic pill followed in fourteen hours by a saline will produce efficient intestinal elimination A glass of hot water, flavored with a dozen drops of lime juice, every hour for six or eight hours, and every two hours thereafter will fulfill requirements of renal elimination (Sugar and alcohol must be omitted, lemon juice is less efficient than lime juice, but may be substituted in four times the quantity Lime juice tablets are more convenient but are inefficacious) The temperature of the room, the hot fluid intake, a hot-water bottle to the feet, and empirin (acetylsalicylic acid) (15 grains or 1 gm) by mouth every third hour for three doses will increase the dermal elimination

For the choking, strangling cough that is so annoying to some patients *opiates should not be given* They check elimination and involve risk of drug habit Two harmless remedies are available for relief of this symptom One is to alkalinize the secretions of the larynx and trachea with sodium bicarbonate, 30 grains (2 gm) every three hours (The physiologist's anxiety about disturbance of the acid base balance is doubtless technically justified, but clinically no harm has ever followed the treatment here advocated) The other remedy is pure honey, a teaspoonful slowly taken from the spoon for the paroxysm, which it usually arrests promptly It is invaluable in children because they take it without protest

Diet should be strictly limited to hot liquids, such as broths and clear soups, including unthickened vegetable soup with plenty of onion juice in it Citrus fruits and other fruits need be limited in quantity only by the particular patient's gastric distress, but a four- or five-hour interval between the intaking of anything in the nature of food is advisable Tea and coffee may be allowed, the quantity and time of taking may well conform to the patient's custom

The foregoing is the initial treatment, duration and repetition depend upon the judgment of the physician in charge In a general way it may be stated that the treatment is continued at a reduced rate after the first day or two The hot soapy bath should be repeated twice daily for a few days, and the other forms of elimination, including a minute daily dose of a laxative, should not be abandoned too soon The best local treatment is silence, the patient writing everything he has to say that is not communicable by a nod or shake of the head, or

other sign If all the elements, general and local, of the treatment mentioned in the foregoing lines be strictly followed, and be not frustrated by the giving of opium derivatives, the patient will be made comfortable and the duration of the disease in almost all cases will be shortened The only likely dissatisfaction will come from patients accustomed by previous use to the effects of harmful and risky medication for such attacks The sulfonamides (q v) may be used if properly safeguarded to minimize the toxic risks, but in a self limited disease practically free from mortality the advisability of their use is for the physician to decide in the particular case Penicillin (q v) is efficacious in some cases

Prognosis, Course, and Termination—The disease is self limited, it runs its course in about ten days if silence be maintained Use of the voice may prolong it With proper care as herein outlined complete recovery may be expected in probably 98 per cent of the cases

Sequelae—Chronic laryngitis may follow lack of treatment and use of the voice In the interval between the acute and the chronic diseases, there may be a *subacute laryngitis*, which under these circumstances is quite properly called a *transitional laryngitis* Its treatment calls urgently for strictest enforcement of the regimen of silence and of the measures mentioned under the heading of "Prophylaxis" in the section "Chronic Laryngitis" Vocal abuse during acute laryngitis is one of the chief causes of ruin of the finest qualities of the singer's voice Myasthenia laryngis is a common sequela in all professional voice users Drug habit may be started by improper medication Chronic bronchitis and bronchiectasis are preventable sequelae, as mentioned in connection with these diseases

ACUTE INFECTIOUS LARYNGITIS IN CHILDREN

Though less frequent in children, acute laryngitis is, in many cases, a much more serious disease than in adults One type, acute laryngotracheobronchitis, will be considered in the section on "Trachea and Bronchi" The factors responsible for the greater seriousness of acute laryngitis in children are anatomic and immunologic The lumen of the larynx is smaller, very little swelling will close it Additionally, the loose areolar tissue filled with lymph spaces is subject to rapidly developing and early edema

Impending asphyxia requiring early and prompt tracheotomy is a much more frequent complication than in adults. Immunity is complete for a short time after an attack of acute infective laryngitis, for a long time there is a partial immunity that lessens the severity of subsequent attacks. The younger child, suffering from his first attack is, in most cases, quite toxic and seriously ill. The subglottic swelling gives a croupy sound to the cough. It is always necessary to exclude diphtheria in the diagnosis, treatment is similar to that of the same disease in the adult.

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ACUTE SPECIFIC LARYNGITIS

Acute specific laryngitis is an acute inflammation of the laryngeal mucosa due to, or as associated with, specific general diseases, or attributable to particular infective agents. These forms of laryngitis are distinct from that definite morbid entity described in the foregoing pages, the acute laryngitis that forms part of the common cold. In measles, scarlatina, pertussis, and variola the laryngeal inflammation is present in most of the cases. In most other acute specific diseases it occurs incidentally.

Etiology.—Pathogenic bacteria are the cause, in some cases the primary factor is the specific organisms or virus causing the general disease, these agents are often followed by secondary invaders. In other instances the infective agent seems to be a specific strain of frequently encountered organisms. Hemolytic streptococci are the most frequent both as primary and secondary invaders. *Streptococcus viridans* is frequently a factor, a hemolytic staphylococcus less often. Other pathogenic bacteria are occasionally found to be causative. Streptococcal

epidemiology is one of the most important developments of modern research. There is much evidence indicating that already present organisms become virulent.

Pathology.—Involvement of the larynx may be by surface infection, extension by continuity, through the lymph channels by septic infarct, or infection of capillary walls by the blood-borne infection. Streptococcemia is a common complication.

Symptoms.—The chief symptom is hoarseness, usually croupy cough is present also, especially in children.

Course and Termination.—The course is usually that of the general or the pharyngeal disease, with which the laryngitis may terminate. It may, however, linger in chronic form. The patient may become a carrier. Complications may prolong the laryngeal disease for months, especially crico-arytenoid arthritis, perichondritis, chondral necrosis, abscess, and stenosis.

Edematous Laryngitis.—This condition is often referred to as a morbid entity. It is, however, a local lesion that may occur in association with any of the many forms of laryngitis. In adults, in a typical case, at the onset the mucosa is seen to be evenly and intensely red with little secretion. As swelling commences and gradually increases the mucosa becomes less and less reddish in color. If the laryngeal edema reaches the stage of occlusion the three rounded masses representing the epiglottis and the two aryepiglottic folds are sometimes pinkish but usually whitish and opalescent or semitranslucent. The surface may be glistening or dull. There may be patches of secretion.

Arthritis of the Crico-arytenoid Joint.—Though in most cases inflammation of this joint is due to extension by continuity from a nearby inflammatory process, or to trauma, it does occur alone or associated with arthritis of the extremities. It is due to causes similar to those of arthritis of other joints, such as focal infection in any region, but particularly foci in the mouth, teeth, and pharyngeal lymphoid tissue. All the controversial opinions of arthritis in general are involved, including so-called "rheumatism." The laryngeal inflammation is limited to the one or to both arytenoid joints. Dull pain or "soreness" are felt on swallowing as the food passes the crico-arytenoid region. The arytenoid eminence may be reddened and swollen, and palpation with the finger or the motion of the passive mobility test will reveal

tenderness early, fixation later. (For diagnosis see under "Paralysis of the Larynx.") Tuberculosis, syphilis, and cancer are always to be excluded before a diagnosis can be made. During the inflammatory stage, treatment is general, not local. Increased elimination by bowels, skin, and kidneys, gently but continuously maintained, is helpful. Eradication of septic foci is of fundamental importance. Ankylosis or, at least, limitation of range of movement is the usual sequela. If unilateral there will be good phonation from compensatory adjustment by the sound side. If obstructive bilateral fixation of the crico-arytenoid joints should remain as a sequela, the joints should be mobilized by external operation (See under operation for midline paralysis, p. 532.)

Acute Specific Diseases—Measles—In this specific infectious laryngitis the laryngeal appearances are usually the same as described in acute laryngitis. In some cases the inflammation is patchy. The secretions at first are thin, watery, and more abundant than in the ordinary type, becoming more purulent, sometimes membranous, toward the end. No local treatment is necessary unless there is stenosis (*q v*), which is rare.¹

Scarlatina—Laryngitis, usually due to specific strain of causative streptococci, is quite common. In most cases recovery is spontaneous, but occasionally septic edema requires tracheotomy. Perichondritis, chondral necrosis, and abscess of the larynx are complications in virulent infections, with stenosis of the larynx as a sequela. All of these conditions are elsewhere herein considered.¹

Pertussis—Acute specific laryngitis and tracheitis are constant features of whooping cough. Submucosal hemorrhages often occur in the larynx, they are not large enough to cause hematoma—only a red ecchymotic spot that darkens. Early and late spots may be present simultaneously. Great relief from choking, strangling paroxysms of cough will be obtained by the system of treatment described for acute infectious laryngitis added to the treatment for the general disease, of course, the management of the laryngeal phases of the case are modified to fit in with the general treatment.¹

Varicella—A specific laryngitis is present as part of the general disease, though the formidable character, present or implied in the name of smallpox, relegates the laryngitis to the

background. The patient would be the better for examination and treatment of the acute laryngitis as hereinafter described.

Typhoid Fever—Laryngitis usually occurs in the third or fourth week. Because of low resistance and secondary pyogenic invading organisms favored by oral sepsis it is prone to end in perichondritis, chondral necrosis, abscess of the larynx, and laryngeal stenosis. These complications require low tracheotomy to prevent asphyxia. This does not immediately arrest the destruction of the devitalized cartilage. Progressive chondral necrosis results in loss of laryngeal framework. Radical local surgery, as applied to similar conditions elsewhere, is contraindicated. After necrosis has ceased and convalescence is well established, core-mold treatment (*q v*) should be used for the stenosis. Prophylaxis is important and consists in best possible oral hygiene, including an antiseptic mouth wash of pure grain alcohol (15 per cent) in warm water. Glycerin added in 25 per cent strength is helpful. The mouthwash is used after each taking of food, it is, of course, not to be swallowed.² The teeth should be gently brushed twice daily. This care, obviously, is proper for any patient, but in typhoid fever the patient seems so ill that the incidental annoyance and fatigue may seem to contraindicate so much oral attention.

Granulocytopenia—In the condition usually called *agranulocytic angina*, and sometimes also called *malignant neutropenia* and *agranulocytic leukopenia*, the ulcerative, necrotic sloughing pharyngeal or tonsillar lesion extends to the larynx. Oftener the laryngeal lesion is in the form of an edema. Local treatment of the larynx is contraindicated. Notwithstanding the contraindication to surgery in granulocytopenia, tracheotomy may be required to prevent asphyxia. The etiology, pathology, diagnosis, and treatment are considered under "Pharyngeal Phases of Blood Dyscrasias."

Infectious Mononucleosis (Glandular Fever)—This disease is usually accompanied with a mild laryngitis that calls for watching, but local treatment is not indicated. Systemic treatment and a general consideration are given under "Pharyngeal Phases of Blood Dyscrasias."

Brucellosis (Brucellosis, Malta Fever, Undulant Fever)—Laryngitis with edema and ulceration complicates this disease in a small percentage of cases. The cause is a milk-borne systemic infection with *Brucella melitensis*. This

organism does not produce a specific lesion in the larynx or pharynx, the local pyogenic infections produce chronic granulomatous lesions objectively resembling tuberculosis, syphilis, or Vincent's angina. The diagnosis is made by exclusion of these diseases (*q 1*) and the positive diagnosis of systemic brucellosis by specialized laboratory technic, cultural and serologic, to demonstrate *Brucella melitensis* in blood and excreta. Prophylaxis is in the avoidance of raw milk, pasteurization affords efficient protection, so does homogenization.

Vincent's Angina—This disease has been called also *trench mouth* since World War I. It is primarily a disease of the pharynx (*q v*), caused by the specific symbiotic organisms (Vincent's spirillum and fusiform bacillus) followed by secondary invaders. The diagnosis is bacteriologic and the laryngeal treatment is given on page 451.

Ludwig's Angina—Abscess of the sublingual space, due to exceptionally virulent strains of streptococci and other organisms always present in the mouth, usually extends to the larynx through the submucous tissues. In a number of the cases we have seen, the larynx was closed by edema though the pharynx was only slightly edematous.

Septic Sore Throat—This not very distinctive title has been applied particularly to a definite type of milk borne streptococcal infection (see under "Septic Pharyngitis," p 150). In some of the cases there is extension to the larynx, resulting in edema, perichondritis, and chondral necrosis, necessitating tracheotomy. The same conditions occur also in conjunction with virulent streptococcal infections not traceable to milk, but the use of raw milk is becoming increasingly risky. Pasteurization affords adequate protection, so does homogenization.

Erysipelas—Invasion of the larynx by the variety of hemolytic streptococcus known as *Streptococcus erysipelatis* is usually secondary to the dermal lesion and is often the result of extension by continuity from a similar lesion in the pharynx (*q 1*).

Influenza—Edematous laryngitis is frequently a feature of this disease. Regardless of what may be the primary infective agent (filtrable virus, *Hemophilus influenzae*, A or B, or other kinds) the laryngeal lesion is usually encountered as typical streptococcal laryngitis. In an epidemic, various members of a family may each have a local complication in a different region—nasal

accessory sinuses, middle ear and mastoid, gastro intestinal tract, larynx, tracheobronchial tree, or lungs. In children under two or three years of age the infection usually takes the form of a distinct morbid entity, an acute laryngotracheobronchitis (*q 1*).

Treatment of Acute Specific Forms of Laryngitis—In practically all cases of the many varieties of acute specific laryngitis described on preceding pages the treatment of the larynx is conservative. Local applications to the pharynx, as elsewhere herein given, should be used, but endolaryngeal applications intended for the destruction or the inhibition of activity of the specific primary infective organisms or secondary invaders usually precipitate swelling and stenosis of the larynx, requiring immediate tracheotomy to prevent asphyxia. It is better to depend upon systemic treatment and pharyngeal applications for the arrest of the specific or the secondary process. Meanwhile the larynx and the neck should be watched so that a low tracheotomy can be done early if the cardinal signs of obstructive laryngeal dyspnea (*q 1*) should develop. During this waiting period the atmosphere of the room should be maintained at 70° F (21° C.) and the humidity at about 50 or 60 per cent, or higher if secretions are thick. The best humidifier is the electric motor type but any little electric water boiler is much less expensive and quite satisfactory for use in the home, in these less severe cases. In the absence of commercial electric current, humidity may be maintained by constant boiling of water, any sort of fuel being utilized for the purpose, if alcohol, kerosene, or gas be used it must not be forgotten that carbon monoxide is deleterious even in small percentage. Bath towels wrung out of boiling water hung around the room may serve as a makeshift. If acute stenosis necessitates tracheotomy the humidification becomes imperative to prevent excessive thickening and crusting of secretions in the trachea and bronchi, and the percentage may well be increased to 80 or 90. Scarification, multiple puncture, and all endolaryngeal surgical procedures, even intubation, are contraindicated in all forms of edema of the larynx. Early, low tracheotomy and rest of the larynx give better results. Systemic treatment is of utmost importance. Chemotherapy has rendered the use of sera obsolete except in case of definitely determined specific organisms like those of diphtheria, in which an antitoxin is specific. With the exception

of penicillin, however, the specific drugs, especially the sulfonamides, are so toxic that their use is contraindicated in mild self-limited infections, and when used must be under strict control as advised in the section on Chemotherapy (p 771). In Vincent's angina, chemotherapy in the form of neoarsphenamine is quite effective when given intravenously as in syphilis. It is well to use it also locally, dissolved (5 per cent) in glycerin, to the pharynx, but this solution should not be allowed to enter the larynx. Bismuth subnitrate should be applied to the pharyngeal lesion in dry powder form four or five times daily.

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ABSCESS OF THE LARYNX

Abscess of the larynx is a collection of pus in the laryngeal tissues. It is not an uncommon lesion.

Etiology.—Infection is always one essential factor in abscess, but for infective invasion there must be destruction of barriers or lowered resistance. Trauma may destroy barriers, systemic disease may lower resistance, either of these factors may enable an infective invasion to get a start behind the barriers. The infective agent most frequently found predominant is one of the hemolytic streptococci, next in frequency is the viridans, but any of the pyogenic bacteria may be present, mixed or predominating. By the time the lesion has reached the stage of abscess it is impossible to determine what the primary infective agent may have been, except

in case of specific infections such as syphilis or tuberculosis. These should be immediately sought in every case.

Pathology.—There are two classes of cases characterized by (1) lesions of the soft tissues and (2) necrosis of cartilage secondary to perichondritis. The soft tissues may be infected through the mucosa, the blood vessels, or the lymph channels. Perichondritis (*q v*) is nearly always the result either of extension by continuity of infected lesions or of direct trauma. Necrosis of cartilage, prolonged healing, and cicatricial stenosis (*q v*) are the pathologic sequence.

Laryngeal Appearances.—A mounding of the tissues covered with inflammatory mucosa is seen in the site of the abscess. If rupture has already occurred an ulcer oozing pus is usually visible at some location on the diminished mound. The ulcer may be covered with an exudate. In case of abscess under the external perichondrium the endolaryngeal examination may show only a swollen inflammatory mucosa, worse on the affected side.

Symptoms.—Productive cough and hoarseness with a thick, muffled tone of voice are usually present. The sputum may be scanty until rupture occurs, it is then purulent and blood streaked. Slight fever present before rupture will subside afterward, unless it is due to associated systemic disease such as typhoid fever or tuberculosis. In extreme toxemia the patient may suffocate without a fight for air. We saw many such cases in typhoid fever.¹

Diagnosis.—In endolaryngeal abscess the reflected and direct appearances are diagnostic. In the extrinsic region, digital examination is helpful. Palpation of the larynx externally is often the best means of diagnosis. There is always thickening and tenderness external to the site of an endolaryngeal abscess. If the abscess is external to the cartilage the boggy sensation under the trained finger is diagnostic. Systemic basic disease if not obvious must be carefully sought by general diagnostic methods, syphilis and tuberculosis are primary diagnostic possibilities, though their presence does not exclude other diseases.

Complications.—Asphyxia from laryngeal obstruction, mediastinitis, multiple abscesses, blood stream infection, chondral necrosis, and cicatricial laryngeal stenosis are the usual complications, the latter always follows unless forestalled by appropriate treatment.

Treatment.—Early and low tracheotomy is the best treatment in cases in which the cardinal signs of obstructive laryngeal dyspnea ($q\ 1$) are present, even in mild degree. If the abscess has not opened spontaneously it may be opened with the laryngeal knife (Fig. 350) used through the direct laryngoscope. Great care and precision are necessary to avoid trauma to the crico-arytenoid joint, the cords, muscles, or the sound perichondrium in any location. Utmost conservatism is essential, the exploration for pus justifiable in general surgery is fraught with disaster in dealing with abscess of the larynx. When a collection of pus is palpable externally it should be approached carefully with the scalpel to avoid incision of normal perichondrium. Here again surgical methods such as cleaning out necrotic cartilage and curettage are contraindicated (See "Perichondritis of the Larynx"). Chemotherapy is important. Methods and dosage are given in the section on specific



Fig. 350—Laryngeal knife for use through the anterior commissure laryngoscope

drugs. When healing is nearly complete the core mold treatment ($q\ 1$) should be begun to prevent stenosis.

Prognosis.—Considered alone the prognosis as to life is good if asphyxia be prevented, and it easily can be if tracheotomy be done early. All nine functions of the larynx can be restored in almost all cases, if there has not been too much loss of laryngeal cartilaginous framework. The duration of suppuration may be rather prolonged under best of treatment.

Sequelae.—Cicatricial laryngeal stenosis follows in almost all cases if not prevented by appropriate treatment (See "Treatment" under "Chronic Cicatricial Stenosis of the Larynx").

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NONINFECTIVE ACUTE LARYNGITIS

Noninfective acute laryngitis signifies acute inflammation of the laryngeal mucosa due to causes other than infection. This refers to the primary condition, any laryngeal lesion may become secondarily infected. Synonyms applicable to many cases are *indirect trauma of the larynx*, *acute traumatic laryngitis*, and *vocal abuse*. The condition is common. Usually it is seen almost every day by the busy laryngologist.

Etiology.—Some form of trauma ($q\ 1$) is the most frequent cause. Shouting, screaming, and excessive use of the voice either in duration or loudness are common forms of indirect trauma. So is cough due to other than laryngeal causes. Since repeal of prohibition, alcohol is an increasingly frequent cause, when combined with shouting, as in carousing and in cheering at football games, it is a potent cause. Direct trauma may be inflicted by foreign bodies. Instrumentation as with the stomach tube or blindly passed bougie, smoke, drugs such as potassium iodide, chemical fumes such as ammonia or chlorine war gases, and many similar causative agents are recorded in our clinical records. Cough due to other than laryngeal causes frequently produces a secondary traumatic laryngitis.

Symptoms.—Hoarseness is the chief symptom. It is usually accompanied by a lowering of the pitch of the conversational voice. A sense of soreness in the throat and pain, usually dull, is felt on speaking (odynophonia).

Diagnosis.—The laryngeal appearances are diagnostic. On examination with the laryngeal mirror the mucosa appears diffusely reddened except the cords, which usually seem pink. If a hematoma is present its deep crimson color will make the cords seem paler. Viewed through the laryngoscope the laryngeal mucosa is diffusely reddened in color. The cords are deep red, not pink. One or both cords may show a rounded tumor-like hematoma ($q\ 1$) or a subepithelial hemorrhage that may be evenly spread or streaked. Questioning will elicit a confirmatory history of the traumatizing episode in the traumatic cases, direct or indirect as the case may be. A patient should always be questioned as to drugs taken, also as to alcoholic beverages, carousing, and shouting, of which they are not proud and seldom make mention.

Complications.—Infective inflammation may follow a primarily noninfective condition.

Treatment.—Silence is the essential and also the only treatment needed except in cases of hematoma on a vocal cord. This always should be opened or ruptured to prevent its developing into an organizing hematoma (*q v*). The procedure is one of great delicacy and precision. If the operator is sure of the accuracy and gentleness of his touch he may use the laryngeal knife (Fig. 350), but in most cases a little superficial pinch with the vocal nodule forceps (Fig. 341) is a safer procedure. Direct laryngoscopy is the only safe method of operative access.¹

Prognosis and Sequelae.—If the cause persists or is often repeated, chronic laryngitis is certain to follow. A singer's voice will be ruined for concert or operatic work, a public speaker's voice will become harsh, and eventually will be lost, the conversational voice will have an unpleasant quality. The treatment just mentioned will obviate all the sequelae, but, unfortunately, strict silence is difficult to enforce.

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DIPHTHERIA OF THE LARYNX

Diphtheria of the larynx is an exudative specific inflammation of the larynx due to the *Corynebacterium diphtheriae*. The pharyngeal, nasal, and systemic phases of the disease have been previously herein considered. Laryngeal involvement as an extension from the pharynx is less common now than before the discovery of the curative power of antitoxin. As a primary and sole lesion it is not rare in children. It may be accompanied or complicated by tracheo-bronchial diphtheria (*q v*).

Etiology.—The infective agent is the diphtheria bacillus (*Corynebacterium diphtheriae*), but the cause of the greater vulnerability of the larynx in some children is unknown. The cause of the severity in laryngeal cases is that the diagnosis is not ordinarily made as early as in the fauces, consequently antitoxin is given late. In the cases in which the larynx has become affected by extension, the cause of laryngeal in-

volvement is late administration of antitoxin for the pharyngeal lesion.

Pathology.—The pathologic processes are the same as those of pharyngeal diphtheria (*q v*). The chief difference is in the loose areolar submucosal tissue of the conus elasticus in children. This tissue swells early, always before membrane appears anywhere, and is the cause of the characteristic croupy cough, and also of the early and severe obstructive dyspnea, so often requiring tracheotomy or intubation.

Direct Laryngoscopy.—Early in the disease the mucosa is intensely red, including the usually pink cords. Below each cord is seen the mounding, swollen subglottic tissue that is seen in all acute laryngeal inflammations in children. Later the intensely red mucosa is seen to have upon it one or more patches of grayish exudate. Usually there is a little blood or a small clot visible at the edge of the patch. When it first appears the exudate may have a watery appearance, but soon it becomes drier, flatter, and more like membrane. At a still later stage there may be a membranous accumulation sufficient nearly to occlude the glottis. In most cases, however, the obstruction is chiefly due to swollen tissue, especially in the subglottic region on each side. In older children a similar condition may be seen in the laryngeal mirror, but reflected examination is rarely satisfactory in a child under the age of about seven or eight years. In adults there is less of the subglottic swelling, and there is less tendency to obstruction of the airway.

Diagnosis.—Those who have seen many diphtheritic larynges through the direct laryngoscope can make the diagnosis by the laryngoscopic appearances and the odor that comes through the tube with the patient's breath. On such a diagnosis antitoxin should be given without delay, even if, as often happens in laryngeal cases, culture from a pharyngeal swab-specimen has been reported to be negative for diphtheria. This, however, does not imply any disparagement of the value of bacteriologic diagnosis, specimens from the pharynx are often negative in laryngobronchial cases. A swab specimen should always be taken from the larynx with a cotton swab introduced into the larynx through the direct laryngoscope, in every case of acute croupy cough in a child, when there is no membrane in the fauces, and it is seldom advisable to wait for the report upon a pharyngeal specimen before doing so. Text.

books recite inferential means of differential diagnosis of laryngeal diphtheria (in the absence of a pharyngeal lesion) from acute laryngitis, laryngismus stridulus, spasm of the larynx, and streptococcal laryngitis. Such an inferential diagnosis may be fatally misleading. Foreign body without any evidence of diphtheria has been found post mortem in children whose death certificates had given diphtheria as the cause of death. Mention of such cases appears in the newspapers. Direct laryngoscopic examination will promptly establish a correct diagnosis and it should never be omitted in any child too young to be satisfactorily examined with the mirror. If the child is so unfortunate as to be so situated that no one capable of doing direct laryngoscopy is available, antitoxin should be given without waiting for a diagnosis

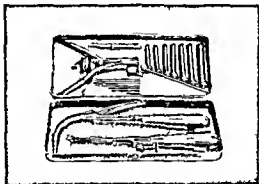


Fig. 351—Instruments for intubation of the larynx for prevention of asphyxia. These are exact copies of the original models perfected by O'Dwyer and never since improved upon.

Treatment.—The general symptoms, diagnosis, and treatment are given in connection with pharyngeal diphtheria. It remains to consider means of maintaining the airway until the disease is cured by antitoxin. The first and most important of these is *aspiration* of secretion and exudate that are occluding the glottic chink, which is much reduced in size by its swollen margins. The silk woven aspirating tube (Figs. 358, 469) is passed through the direct laryngoscope (see "Aspiration"). The tube must be used gently, and no swabbing is permissible. Done in this way there is no local reaction and intubation is obviated in many cases.

There are two methods of maintenance of airway: intubation and tracheotomy. The indication for either method is the appearance of the signs of obstructive laryngeal dyspnea

(qv). Cyanosis should not be awaited, the color is oftener ashy gray than blue. The child will be restless if not extremely toxic or drugged with sedatives. Efficient direct aspiration will often obviate necessity for either intubation or tracheotomy.

For diphtheria in children *intubation of the larynx* is a satisfactory procedure, provided the patient is in a hospital where there is a constantly and immediately available trained intubator who can promptly replace the tube in case it should be spontaneously expelled, and also provided the child is not too feeble from toxemia. A profoundly toxic child exhausted from loss of sleep, a long fight for air, and prolonged anoxemia, with respiratory centers fatigued from continuous high carbonic-acid tension, will be too feeble to cough out any membrane that may be pushed down ahead of the intubation tube. He is likely to give up, stop breathing, and die of asphyxia and cardiac cessation at the first attempt at intubation, and the risk is greater if the operator is not facile. On the other hand, the procedure of *tracheotomy* not only adds nothing to the danger in such a case, but on the contrary it is directly stimulating. Moreover, the tracheotomic wound affords access for promptly clearing the trachea and for instant administration of oxygen below the laryngeal obstruction. This can be done even if respiratory movements should cease. A few movements of artificial respiration will draw air into the lungs through a tracheotomic wound, but not through a more or less obstructed intubation tube. Of course, neither intubation nor tracheotomy should be postponed until the child has reached such a dangerous condition, but as a clinical fact, for one reason or another, both these procedures are almost always done later than they should be done. One reason is the delay awaiting action of the antitoxin in the hope that it will render the procedures unnecessary, but even in the days before diphtheritic antitoxin was known the child was usually almost moribund before operative relief was undertaken.

Technic of Indirect Intubation of the Larynx

—The necessary instruments are shown in Figure 351. They are those of O'Dwyer, in a half century of use they have never been improved upon in design or material. One important piece of equipment is not shown, namely, a tank of oxygen filled with 7 per cent carbon dioxide admixture. Sterile petrolatum

and sterile gauze sponges should be ready. Tracheotomy instruments should be sterile and ready for emergency.

The child should be flat on his back with a small and low pillow under his head which is about 2 feet (60 cm.) distant (toward the foot of the bed) from the head board. The nurse holds the child's head with the nose toward the zenith and maintains the mouthgag in position. The patient's forearms are held down on the bed by any capable person, the knees being restrained by the person's right arm and axilla (Fig. 352). The child's body is lengthwise parallel to the edge of the bed. The opera-

ting the laryngeal orifice the axis of the tube in line with the lumen of the larynx. The tube is then released by the right thumb pushing forward on the detent piece. The tube is gently pushed on down into place with the left index finger and the intubator is withdrawn. The tethering loop is retained on the fourth finger and is watched for a few minutes. If it slowly disappears in the patient's mouth the tube has missed the larynx and is being swallowed; it must be withdrawn with the tether. If it does not disappear the end of the tether is attached to the side of the patient's neck with a bit of adhesive plaster. The patient must be watched to prevent his pulling the tube out by grasping the tether.



Fig. 352.—Intubation of the larynx. The child's head is supported and controlled by the chief nurse whose left arm passes under the child's neck, the index of the left hand holding the bite block which props the child's mouth open. The assistant nurse holds the child's forearms down on the bed; if necessary she drops her elbows on the child's knees. The operator has the tip of his left index finger in contact with the patient's arytenoids. The operator's right hand holds the intubator with thumb on the detent and the silk loop on his little finger. The tube is slid in along his left index finger until its distal end almost reaches the arytenoids. The tip of the tube is inserted between and anterior to the arytenoids by a combined movement of dragging anteriorly and raising the handle of the intubator (Fig. 353). If the dragging or raising be omitted the tube will go into the hypopharynx and be swallowed until checked by the silk loop. When the tube is felt to be properly placed it is pushed off with the detent and the intubator is withdrawn.

tor loops the silk tether over his little finger, and holds the intubator in his right hand, which is touching the chest of the patient. The index finger of the left hand is passed back over the tongue until the epiglottis is felt. The epiglottis is drawn forward to expose the laryngeal orifice. The right hand then guides the tube back along side the left index finger until the distal end of the tube reaches the left finger tip which is holding the epiglottis forward against the back of the tongue. The shaft of the intubator, which until now has been held parallel to the chest wall, is raised to a vertical position (Fig. 353). Now the most important movement in the whole procedure is executed. The handle in this high position is pulled anteriorly. This drags the tube into a position overhang-

The foregoing description is of indirect intubation in the recumbent position. The procedure is often described and done, with the child in the upright position, but the mortality in such position is much higher. It is inviting disaster to place a child, whose heart muscle and respiratory centers are nearly paralyzed by the toxemia of diphtheria, in the upright position. To make matters worse the child's chest is usually bound up tightly to restrain his arms still further embarrassing respiration and heart

action. Furthermore, if necessity for tracheotomy and artificial respiration is precipitated by attempt at intubation, as it often is, potentially lifesaving moments are lost in placing the child in another position recumbent on the bed. In our opinion the upright position will be abandoned for intubation in diphtheria, though doubtless in the same reluctant way as it was for tonsillectomy under general anesthesia, and for the same good reason, namely, higher mortality.

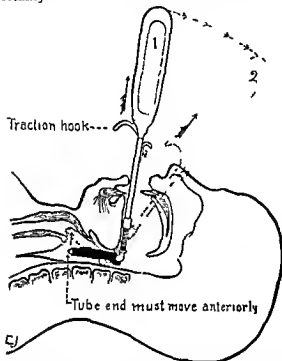


Fig 353—Sketch showing schematically the most important point in blind intubation of the larynx. When the distal end of the intubation tube has reached the arytenoids the distal end must be both pulled and pointed anteriorly to enter the larynx. The laryngeal orifice is anterior to the pharynx and under the base of the tongue.

When all is going well the silk tether should be cut and removed, making sure that the tube is not thereby withdrawn, counterpressure with the tip of the left index finger will prevent this accident. The tether loop should be cut on only one side of the knot and the traction should be made on the knotted end; the end without the knot will thus come through the eyelet, whereas the knotted end would stop at the knot. The trained ear will detect a peculiar tubular sound of the breathing when the tube is clear. The presence of partially obstructive secretions will alter the note. There is some coughing at first, and it also has a peculiar sound because of lack of cordal cooperation in the bechic cycle, but cough soon becomes only occasional.

Dehydration must be prevented. After an hour or two of rest the child should be given good cool water to drink. If it cannot be taken

without strangling cough from its intrusion into the larynx, both water and liquid food must be given at regular intervals through a soft rubber catheter passed into the esophagus through the mouth or nose, usually the latter.

The after-care consists in preparation for both immediate reintubation and tracheotomy, prevention of dehydration, maintenance of room temperature at 72° F, with a humidity percentage of about 70, and giving the child rest. More than absolutely essential attention is injuriously fatiguing.

Pitfalls in Indirect Intubation of the Larynx—

In all purely manual things, knowing how to do them is not enough, facility comes only from nerve cell habit created by going through precisely the same sequence of movements many times. In rehearsal of the sequence of movements necessary for gentle facile intubation a large, soft rag doll, such as any nurse could make, would serve perfectly. In manual rehearsal of intubation the following axioms should be memorized by repetition.

- 1 Select the proper size tube for the age of the child according to the scale supplied with the instruments.

- 2 Cut off a piece of braided silk about 2 feet (60 cm) long, thread it through the eyelet in the upper end of the tube, tie the two ends of the silk together to form a tether, remember to keep this knot close to the little finger during intubation so the silk string can be cut at one side of it.

- 3 Lubricate the surface of the tube thinly with a bit of gauze dipped in sterile liquid petrolatum.

- 4 Observe strictly the successive positions of instrument, tube, and fingers, as given in Figures 352 and 353, and go through them in exact sequence two or three hundred times every day for a week.

- 5 Keep always in mind the forward pull with the handle high and rising up toward the forehead of the patient. This pull, in this position, prevents the tube going posteriorly into the hypopharynx on its way to the esophagus.

- 6 Feel the orifice of the larynx before releasing the tube; you can recognize it as two small firm knobs, back of the epiglottis. The orifice of the larynx is between the epiglottis and the little knobs (Fig 353).

- 7 Push the detent at the same moment the left index finger seats the tube in place in the larynx.

- 8 Cut the silk string at one side of the knot, and pull on the knotted end while making counterpressure on the tube with the left index finger to prevent withdrawal of the tube.

In connection with intubation the following axioms will call attention to errors that should be avoided: (a) Do not postpone intubation until the child is worn out with toxemia, anoxemia, and the fatigue of fighting for air. (b) Do not use force enough to penetrate tissue,

there is little resistance to the tube when it is in the orifice of the larynx (c) If you cannot introduce the tube, do not substitute a smaller size, unless the child is extremely small for his age, and even then do not risk more than one size smaller (d) Do not persist until breathing has ceased Do a tracheotomy (e) Do not attempt intubation without having laid out a full set of instruments for tracheotomy, sterile and in readiness for instant use An oxygen tank is necessary, an anesthetic is not, for emergency tracheotomy (f) Watch for nasal obstruction, it interferes greatly with respiration in children It may or may not be diphtheritic It may be so-called "fibrinous rhinitis," "croupous rhinitis," or "membranous rhinitis," or simply an accumulation of mucous, more or less dried, viscid or purulent A few drops of warm isotonic salt solution, instilled into the nostrils when needed, will promote clearance

Extubation—Usually when the swelling has subsided so that the tube is no longer needed it will be coughed out, or coughed up and swallowed If not, extubation should be done on the fifth or sixth day Before doing it a fresh duplicate tube should be sterile and strung in readiness for reintubation if necessary Tracheotomy instruments should also be ready The extubator (Fig 351) may be used under finger guidance if the procedure is well practiced beforehand Otherwise trauma may be inflicted A simpler and better way is to squeeze the tube up into the pharynx where it can be caught between the index and middle finger of the operator's left hand The squeezing up is done very gently with the thumb and first finger of the right hand applied externally on the trachea just below the lower end of the intubation tube, which location is easily detected by the soft compressibility of the tracheal rings unsupported by the tube The child's head should be slightly extended to bring the trachea forward into prominence No trauma will be inflicted if the squeezing is gently done The position of the child on the bed and the holding of the bite block are the same as for intubation

A swallowed intubation tube will pass harmlessly through the gastro intestinal canal, but in case of a missing tube it should never be taken for granted that this has happened A roentgen ray examination must always be made to be certain the tube is not somewhere in the patient Such an examination may await discharge of the patient from quarantine, or

isolation, but the sooner it is done the better In one case a child was sent to bronchoscopic clinic for removal of an intubation tube from the right bronchus¹

Direct Intubation of the Larynx—The direct method has lifesaving advantages (1) It gives absolute control of the airway This is fully appreciated by everyone who has had much to do with children (2) Obstructive secretions can be aspirated before insertion of the intubation tube (3) This aspiration may entirely obviate the necessity of intubation (4) If respiration should cease a small bronchoscope can be instantly inserted and oxygen gently insufflated into the tracheobronchial tree This procedure usually promotes breathing at once, if it does not, alternate raising of the child's elbows over the head, bringing them back again with slight pressure on the thorax, in Sylvester's method, will pump into the lungs the oxygen that is flowing into the bronchoscope through its side inlet, with which all bronchoscopes are made (Fig 449) (5) If it be decided that tracheotomy is preferable to intubation an orderly low tracheotomy can be done with a bronchoscope in the trachea (q)

For direct intubation the O'Dwyer intubation tube is used, the size being selected as for the indirect procedure It is held with Mosher's intubator The laryngoscope must be large enough in lumen to permit passage of the required size of intubation tube through it, unless the operator has acquired the knack of passing the tube alongside of the laryngoscope, which is really the best way The technique of exposure of the larynx for direct intubation is the same as shown in Figure 342. The next step is the aspiration of secretions and membrane (Figs 469, 470) This may give such great relief that intubation may not be necessary If, however, the indrawing does not promptly cease, the intubation tube should be slipped in (Figs 354, 355) A string is not necessary but may be used if deemed advisable Its only use would be to enable the nurse to withdraw the tube if it should become dangerously obstructed

Direct extubation is easily done with the laryngeal forceps, (Fig 341) by expanding the jaws inside the proximal end of the tube, after exposure with the direct laryngoscope, but the squeezing up method of extubation may be used In either case necessity for reintubation with a duplicate fresh tube must be borne in mind as a possibility

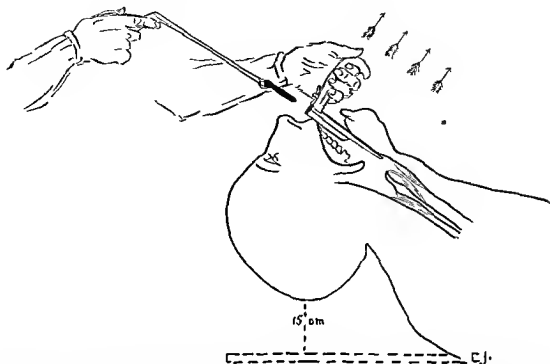


Fig. 354 —The larynx is exposed to view with the laryngoscope (Fig. 34), using a size large enough to permit the selected intubation tube to pass through it. After aspiration of the larynx and trachea with the silk woven aspirating tube the intubation tube is poised as here shown while the operator carefully makes sure the larynx is properly exposed with the laryngotracheal axis in line (see Fig. 355)

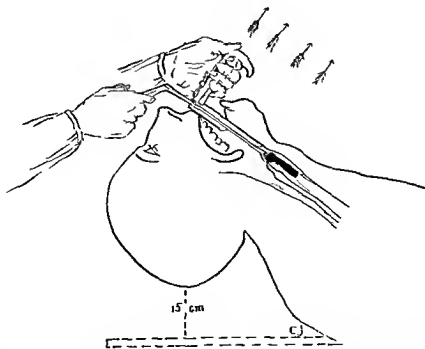


Fig. 355 —Guided by the eye the tube is passed into the larynx, and seated in place, with supraglottic overhang posteriorly between the arytenoids. It is then released and the intubator is withdrawn. If the intubator is properly opened, yet the tube is not firmly enough held for retention by the infraglottic swell to prevent it coming out with the intubation tube, the next size larger tube is needed.

Complications.—Impending asphyxia requiring tracheotomy is the most frequent complication, but it, like intubation, is much less frequent since direct laryngoscopic aspiration has come into general use. *Pulmonary atelectasis* from valvular bronchial obstruction is not infrequent in overlooked tracheobronchial diphtheria in which there is no membrane in the fauces or pharynx. If bilateral it is promptly fatal. The condition called *massive collapse of the lungs* was first discovered at autopsy in patients who died of asphyxia as a terminal phase of diphtheria. Occurring thus, it was naturally attributed to paralysis. It has since

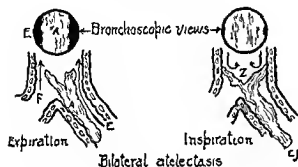


Fig 356.—Schematic illustration of the mechanism by which a flapping mass of cast off diphtheritic membrane produced bilateral atelectasis in a child aged three years, as seen bronchoscopically during artificial respiration. On artificial expiration, spaces for air opened at E, F. At the beginning of inspiration the membranes flapped outward, checking inflow as shown by the bent darts Z. The mechanism had evidently produced a fatal atelectasis; an intense cyanosis of dermal and mucosal surfaces indicated that the heart had been working after respiration had ceased and autopsy showed both lungs completely collapsed. The membrane was found in the expiratory position shown here. In the circles the corresponding bronchoscopic appearances are schematically presented. The bronchoscopy was done after death. The child was dead on arrival at the accident ward.

been observed,² however, that in tracheobronchial diphtheria a check-valve mechanism can pump out the air from both lungs causing bilateral atelectasis and death (Fig 356). Direct laryngoscopic aspiration used early and repeated as often as indicated will prevent such a disaster.

Prognosis.—The prognosis of laryngotracheobronchial diphtheria is not bad if the diagnosis is made early and when direct laryngoscopic aspiration is added to early use of antitoxin. Lacking these the prognosis is extremely unfavorable.

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NOCTURNAL DYSPNEIC ATTACKS IN CHILDREN

Nocturnal dyspneic attacks may be defined as sudden dyspneic seizures occurring after an hour or more of sleep.

Much confusion and vagueness abounds in the literature, and consequently in the minds of practitioners, concerning this syndrome, largely because various authors are not writing about the same thing, or are grouping consideration of different morbid entities. In all of these entities the child, after a period of sleep, suddenly awakens with intense dyspnea almost always inspiratory.

Inflammatory Nocturnal Laryngeal Dyspnea.—Acute laryngitis from various specific infections, such as various streptococci and staphylococci, *Hemophilus influenzae*, and *Corynebacterium diphtheriae*, sometimes sets in suddenly during the night with an attack of dyspnea and croupy cough in children. The prodromal symptoms are sometimes absent or so slight as to have been overlooked. These inflammatory diseases involving the larynx and sometimes producing the nocturnal dyspneic syndrome are separately, elsewhere herein described. *Asthma* is associated with nocturnal attacks of dyspnea, but the dyspnea, not being laryngeal, is expiratory in character, and it also is considered elsewhere (see under "Bronchoscopy in Relation to Asthma" in Part V).

Noninflammatory Nocturnal Dyspnea.—In the absence of laryngeal inflammation the nocturnal dyspneic syndrome occurs in two types of case, namely, in normal children as a physiologic reflex, and as a distinct morbid entity known as "laryngismus stridulus," also called "spasmodic croup."

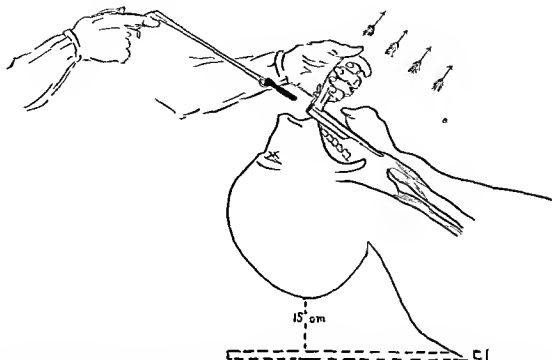


Fig. 354—The larynx is exposed to view with the laryngoscope (Fig. 34) using a size large enough to permit the selected intubation tube to pass through it. After aspiration of the larynx and trachea with the silk woven aspirating tube the intubation tube is poised as here shown while the operator carefully makes sure the larynx is properly exposed with the laryngotracheal axis in line (see Fig. 355)

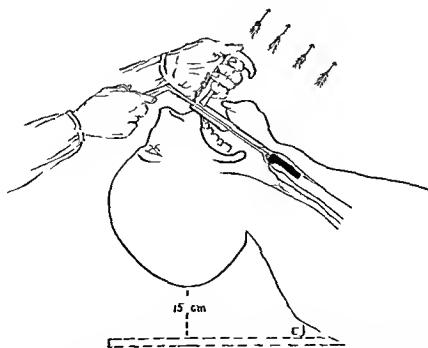


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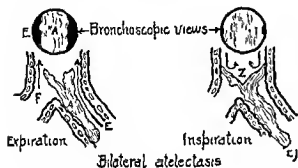


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Noninflammatory Nocturnal Dyspnea.—In the absence of laryngeal inflammation the nocturnal dyspneic syndrome occurs in two types of case, namely, in normal children as a physiologic reflex, and as a distinct morbid entity known as "laryngismus stridulus," also called "spasmodic croup."

Nocturnal Dyspnea In Normal Children—A dyspneic attack of sudden onset occurs a number of times in the life of every child from the overflow of pharyngeal secretions into the larynx during sleep in a position more or less dorsal. Normally these secretions drain away through the tonically closed esophagus by capillarity, and occasionally there is an overflow into the larynx. The effect of this intrusion is a sudden and violent exhibition of activity by the cough reflex which has been called "the watch dog of the lungs."¹ The first effect of the intrusion is a snapping shut of the glottis. The effect of this is to produce air hunger. Attempt to inspire carries some of the secretions below the glottis. A violent tussive attempt to expel the intruder expels residual air. The increasing air hunger is accompanied by an intensely alarming sense of suffocation. The violent inspiratory effort, making pressure on the inclined floor of the ventricles, tightly closes the glottis (Fig. 357). The child is now thoroughly awake but frightened. The glottic closure soon yields to the pull of the posticus muscles, the glottis opens, air enters, and the "watchdog" activities are completed with the expulsion of the intruder by the cough reflex. The paroxysm of coughing soon ends, having a somewhat hoarse and croupy sound toward the last. The child goes to sleep and by morning all hoarseness has disappeared. Rarely there are subsequent attacks before morning, there may be none for weeks or months. Most observant persons can recall such attacks during childhood or later in life. The attacks are more likely to occur, as well as being more frequent and intense, in children with adenoids because nasal obstruction increases dyspnea and increases the accumulation of secretion in the pharynx ready for dumping into the larynx. Hypertrophic tonsils decrease the size of the reservoir so that less secretion is required to reach the overflow level, and they increase dyspnea by their bulk which diminishes the available airway. These are pathologic conditions but the attacks can and do occur without their aid, and the attacks are essentially normal activities of the protective mechanism for stopping and expelling intruders that otherwise would reach the lower air passages. When these attacks occur in older children and in adults they are less severe and alarming because the passages being larger and the intellectual faculties being more developed, the

sense of suffocation is not so terrifying, there is faith in the temporary character of the air hunger. If the larynx of an infant, older child, or adult be examined as soon as the paroxysm has subsided it will be noted that the only abnormality is vascular engorgement of the mucosa. If examined the next day the mucosa will be found to be normal, in typical cases.

In the condition mentioned in the preceding paragraphs the secretions in a mechanical sense may be considered as an endogenous foreign body. An *exogenous foreign body* may produce the same kind of attack. The differential diagnosis is given in connection with the general subject of foreign body in the air and food passages.

Laryngismus Stridulus (Spasmodic Croup)—This is a definite morbid entity which should not be confused with any other condition.² There is often an hereditary factor in the susceptibility of some children between the ages of two and six years. It is usually associated with abnormal calcium metabolism. Without noticeable prodromes, the child after a few hours of peaceful sleep awakens with a peculiar hollow, croupy cough, differing in quality from that heard in any other disease, even true croup (diphtheria). The cough is quickly followed by a crowing inspiration and then by the typical inspiratory obstructive laryngeal dyspnea. In his all too active fight for air the child sits up and may try to clutch at the bedpost for support. His eyes stare wildly and his expression shows the terror of suffocation. The skin is cyanotic, the nails and lips are bluish. After an hour or two of lessening cough, hoarseness, and general distress the attack subsides and the worn out child falls asleep. If the larynx be examined at the end of the attack only vascular engorgement of the mucosa is evident. Milder attacks occur, especially in older children, either primarily or as the severity decreases with or before the approach of the age limit. Direct laryngoscopic oxygen insufflation (*qv*) is the best treatment, if it were always available, tracheotomy, rarely required anyway, would never be necessary. The next best thing is to take the child to a warm room and have the mother smilingly pat the child and quiet his fears. If the mother expresses the terror she feels it naturally increases the terror of the child. Though the room should be warm it should not be what is usually called "stuffy," and steam inhalations should be immediately started.

Means should be always in readiness when a child in the family is subject to attacks of laryngismus stridulus. Between attacks everything should be done to put the child in best possible state of general health. Calcium metabolism and endocrine disorders usually require attention. An excess of vitamins should be constantly maintained. The prognosis is good both as to recovery from the attacks and as to their cessation after the sixth year, if not before.¹

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CHRONIC LARYNGITIS

The term "chronic laryngitis" signifies a chronic inflammatory disease of the laryngeal mucosa. So much confusion has resulted from adding names of secondary and other lesions to the word "laryngitis" that it seems best to describe first the basic disease.

Etiology.—Probably no one in temperate zones reaches maturity without having had a chronic laryngitis. Repeated infection and trauma, including indirect as well as direct forms, are the chief classes of exciting causes. Infections may be exogenous or endogenous, the latter source including the paranasal sinuses and pharyngeal lymphoid tissue. Vocal abuse is the commonest form of indirect trauma (q.v.). Tobacco smoke and air-borne dust are the chief forms of direct trauma. Mouth breathing contributes by loss of nasal filtration and humidification of inspired air. Systemic causes are constant vasodilatation of the laryngeal mucosa with alcohol, in adults, avitaminosis and malnutrition from lack of food or from unbalanced diet in children.

Pathology.—Diffuse hyperemia, round-cell, and fibrotic changes are to be noted in the mucosa and submucosa. The mucosal glandular elements show increased activity early, atrophic changes later.

Reflected Appearances.—The mucosa is dif-

fusely reddened, the tone of the red being dull instead of bright. In gaging tone of color the degree of illumination is a large factor, it should always be the same. The redness of long standing chronic laryngitis is patchy. The illusion of pearly luster of the reflected image of the cords is absent, the color is pinkish early, grayish later, but in either stage the surface appears slightly roughened, dull, and lusterless. The cords lack the illusion of thinness, flatness, and sharpness that characterizes the normal reflected image, but at the early stage the edges appear only slightly rounded. The so called vocal process appears whitish early, in contrast with the deeper tone of the cord, but later the contrast is not noticeable. Enlarged capillaries, though not so conspicuous as on the epiglottis, may be visible on the cords, affording contrast where they pass under a pellet of surface secretion. Stringiness of secretion is a conspicuous feature of the laryngeal image in chronic laryngitis. On inspiration after phonation a string of sticky secretion may be seen stretching across the glottis from one cord to the other. During phonation the vibration of the cords may be interfered with by a pellet between the cords or by a pellet loading a cord like a damper, in either case producing a hoarseness or roughness of voice, often a change of pitch or a break in the tone. From habit the patient clears his throat by a semitussive blast that is usually spelled "h-e-m" or "a-h-e-m." The habit has become so general and so fixed, as preparatory to speech, and chronic laryngitis is so prevalent, that this sound has become accepted into the language as an interjectional word to attract attention or to request hearers to listen to the speaker.

Appearances on Direct Laryngoscopy.—Looking directly at the mucosa with a light adjustment always the same, the appearances of the chronically inflamed mucosa are obvious to anyone familiar with appearances of mucosal surfaces in health and disease. The redness of the cords, the roughening of the epithelium, the enlarged cordal vessels, the rounded edges of the cords—all these inflammatory changes are conspicuous and typical. Additionally there is apparent a roughened condition of the surface brought out conspicuously by the oblique illumination afforded by the one-sided position of the little lamp in the distal end of the laryngoscope. This roughening shows two distinct features: (1) furring of the epithelium all over

the mucosal surface, and (2) a granular condition limited to certain areas. The furring is especially noticeable on the cords, which explains the dull grayish appearance so often seen with the mirror, in chronic laryngitis. The granules are minute elevations due to enlarged mucosal glands. They are most numerous over the areas where glands are plentiful, in the ventricles, on the ventricular bands, and arytenoid eminences. They are especially to be noted on the floor of the ventricle when brought into view by raising the ventricular band with the lip of the tube mouth. There are no ulcers, but the oblique illumination of the laryngoscope brings out clearly patches of erosion of the epithelium that are not seen by the vertical ray of the mirror, just as an otherwise invisible shallow corneal erosion is rendered visible by oblique illumination.

Symptoms.—In almost all cases the chief complaint is vocal impairment, usually hoarseness. Commonly it is intermittent, worse in the morning after a night's sleep and in the evening after excessive use of the voice all day. On questioning, additional symptoms are elicited, particularly local discomfort, a feeling of swelling, an aching, and a tickling sensation causing cough. In all cases there is a necessity for clearing the voice before speaking. This last may have become so habitual that the patient is unaware of it. Professional voice users get to know that they must never start to talk or sing professionally without first clearing out secretion. It is removed from the cords, though they do not know that. The amount of secretion is small and it may be in the form of little pellets of jelly-like, "short," "moss-agate" (qv) tracheal secretion.¹

Diagnosis.—The appearances in the mirror and through the direct laryngoscope are typical, but no diagnosis of chronic laryngitis should ever be made without having seen all of the mucosal surface of the larynx. The floor and roof of the ventricle are invisible in the mirror, and so is the anterior commissure in many patients because of an overhanging epiglottitis (Fig. 345). Cancer must always be the first diagnostic possibility to be excluded before a diagnosis of chronic laryngitis is made, tuberculosis comes next and myasthenia laryngis third. In all three of these conditions, chronic laryngitis is commonly present, their diagnosis and differential diagnosis are considered under their respective headings.

Varieties of the Disease.—Laryngeal literature contains a confusing number of names coupled with the word "laryngitis" some of which are morbid entities, others not. *Laryngopharyngitis* is applicable when both the indicated regions are involved, if the pathologic conditions in each are the same, often they are not. *Hemorrhagic laryngitis* may be a purpuric or an *ecchymotic* laryngitis or a *hemoptysis* with streaks of blood seen in the larynx but possibly from another source. *Ulcerative laryngitis* is not a distinctive term, it might be applicable to any kind of ulcer, syphilitic, tuberculous, cancerous, or traumatic, for example. *Medicamentous laryngitis* occurs from administration of drugs, usually in idiosyncratic individuals, we have seen cases due to potassium iodide, arsenic, mercury, bismuth, lead, vegetable drugs, amido-pyrine, dinitrophenol, the sulfonamides and others of the benzene group.¹ *Exudative laryngitis* includes any of the many laryngeal diseases in which a liquid, semisolid, or solid exudate is deposited in or on any laryngeal tissue. *Dermatopathic laryngitis* has been noted in scleroderma, pemphigus, impetigo, lichen ruber planus, and xanthoma.¹ *Herpetic laryngitis* may be associated with dermal, labial, or ophthalmic herpes, or may occur alone, it is ephemeral and requires no treatment.¹ *Phlegmonous laryngitis*, like most other diseased conditions called "phlegmonous," is rather loosely applied, it may mean abscess of the larynx, perichondritis, chondral necrosis, or any severe submucosal inflammation of the connective tissue. *Edematous laryngitis* is a term that has been loosely applied to many different forms of laryngeal inflammation as well as to numerous noninflammatory edemas to which the suffix -itis is obviously inapplicable. *Catarrhal laryngitis* is a loose and obsolete term dating from Hippocrates, it becomes absurd when modified in the contradictory term "dry catarrh." *Allergic laryngitis* means the participation of the larynx in the general mucosal reaction of hay fever and asthma, in some of the cases it is largely secondary to the trauma of cough (for treatment see under "Allergy of the Nose and Paranasal Sinuses, and Related Conditions"). *Glandular laryngitis* is the name that was given to an inflammatory condition assumed to be primary in, or chiefly affecting, the glands of the mucosa, such limitation has not been demonstrated histologically. *Nodular laryngitis* implies a distinctive kind of laryngitis as-

sociated with vocal nodules, these benign tumors, however, are often organized hematomas unassociated with a chronic laryngitis. *Croupous laryngitis* is a name formerly applied to any case in which there was a croupy cough. *Symptomatic laryngitis* is a general term that has been applied to laryngitis occurring as one of the symptoms of local or general diseases such as the exanthemas, typhoid fever, diphtheria, and influenza, whether the laryngeal condition was specific or not.¹

Complications.—The most common complication is chronic tracheitis. The building up of masses of round cell infiltration that becomes fibrotic leads to benign tumor formation, overworked muscles, crico arytenoid arthritis, and myasthenia laryngis. The impaired tone leads to forcing the voice with consequent submucosal hemorrhage and organizing hematoma. Habitual forcing of the voice leads to fibrosis, a vicious circle, and glandular atrophy.¹

Prophylaxis.—Tobacco smoke, alcohol as a beverage, a dusty atmosphere, vocal abuse, and other causes should be eliminated. Proper care of attacks of acute laryngitis to avoid a residual subacute laryngitis is of utmost importance in prophylaxis of the chronic laryngitis. Absolute silence during the acute attack and convalescence is essential. Oral hygiene as mentioned under "Treatment" is important prophylactically.¹

Treatment.—Search for and elimination of the causative factors are the first steps. Nasal stenosis and sinus and tonsillar disease are frequently-found factors. Trickling of pharyngeal secretions into the larynx during sleep should be minimized by a low pillow and sleeping on the side rather than on the back. The continuous maintenance of the best possible oral hygiene is helpful. The teeth should be cleaned after each meal and again the last thing before going to bed. Vocal abuse should be stopped, if possible, the best means is an enforced regimen of silence (see under "Myasthenia Laryngis"). Complete and thorough general medical examination should be made to determine need of general treatment. Search should be made for a focus of infection elsewhere, whether one is or is not found in nose, throat, ear, or teeth. If one is found, proper treatment may benefit the laryngitis. A warm, moist seashore climate is beneficial. High and dry climatic conditions so helpful in pulmonary tuberculosis are not favorable for a patient with nontuberculous

chronic laryngitis. Local treatment by silver nitrate will cause or perpetuate a chronic laryngeal inflammation, other metallic salts are likewise injurious. The best local application is a few drops of a 1 per cent solution of Mono-P-Chlorophenol dropped into the larynx thrice weekly with a laryngeal syringe (Fig. 341), under guidance of the mirror.

Prognosis and Sequelae.—If the patient will obey orders, perfect restoration of laryngeal functions may be expected. There is no direct danger to life.

Permanently impaired voice is certain to follow prolonged chronic laryngitis. Like any other chronic inflammatory lesion subject to constant irritation, chronic laryngitis may favor cancer of the larynx. As a clinical fact the sequence is common. Prolonged severe chronic laryngitis ends in hypertrophic laryngitis, the sequela of which is, eventually, atrophic laryngitis or laryngitis sicca. Chronic tracheitis, with mossy agate tracheal sputum, is a common sequela as well as an associated disease.¹

Chronic Hypertrophic Laryngitis (Hyperplastic Laryngitis).—This is a prolonged and severe form of chronic laryngitis in which years of chronic inflammation have built up a *fibrosis of the larynx*. The mucosa and submucosa are thickened. Usually the thickening is evenly diffused over the whole larynx. In all its clinical features it is similar to the just described chronic laryngitis except for the results of long duration which entails (1) a greater degree of pathologic change, (2) more hoarseness, (3) a coarse rasping voice, (4) longer duration of treatment, and (5) a less favorable prognosis as to restoration of function, though not as to life.

Chronic Polypoid Corditis.—This is a localized type of hypertrophic laryngitis in which the hypertrophy takes the form of spindle-shaped, edematous masses symmetrically arranged on the anterior half of both cords, in some cases extending backward to the vocal process. Histologic study shows an edematous structure with slight fibrosis similar to that seen in the edematous nasal polyp. The treatment is the same as in chronic laryngitis, with the addition of the operation of scalping off the edematous tissue. This usually required procedure calls for utmost care to avoid damage to normal basal tissues, as in removal of vocal nodules (q.v.). It should be stated that the mode of origin of the latter is entirely different, though

the operative treatment and the pitfalls are the same

Chronic Subglottic Laryngitis—This is a form of hyperplastic laryngitis that is limited to the subglottic region. The building up of hyperplasia in this region ends in cicatricial contraction and a fibrous stenosis of the *conus elasticus*. The stricture usually yields to direct laryngoscopic bougienage. In the rare cases that do not yield to this method core mold treatment will be successful.¹ Both of these methods are described under Chronic Cicatricial Stenosis of the Larynx.

Myositis of the Larynx—Any one or more of the laryngeal muscles may be the site of acute or chronic inflammation, as (a) a deep penetration of a mucosal laryngitis, (b) extension of a connective tissue lesion, (c) part of a systemic myositis, (d) an independent condition. As a primary lesion, myositis of the larynx may be due to (1) direct or indirect trauma (q v), (2) infection, pyogenic or specific, (3) infestation with *Trichinella spiralis*. Whatever the cause, the chief symptoms are hoarseness and dull aching pain in Adam's apple, the pain becoming worse on phonation. Trichiniasis as a cause is inferred from a systemic diagnosis. No efficacious treatment is known for it. Other forms require systemic treatment with vocal rest and the external application of dry heat of as high degree as can be borne.

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CHRONIC ATROPHIC LARYNGITIS

Chronic atrophic laryngitis is a form of chronic laryngitis in which atrophy has followed fibrosis. In the earlier stages it constitutes a *laryngitis sicca* in its later stages it has been called *ozena*, though this name has been applied to a supposed specific disease. It may occur independently or in association with

atrophic rhinitis, or with atrophic tracheobronchitis, rarely all of these forms may occur in the same patient. It is not an uncommon affection.

Etiology and Pathology—Chronic atrophic laryngitis is the result of a hypertrophic laryngitis that has gradually built up, during years of chronic inflammation, a thick accumulation of fibrous tissue in and beneath the mucosa. Many of the mucosal glands are destroyed by the fibrotic contraction. For want of their secretion the mucosa is dry, rough, and dull in appearance. Long continuation of the process results in total destruction of the serous elements in the mucus producing glands and what secretion remains is thick and tenacious, it dries into crusts. The adherent crusts in time become detached, the irritated underlying mucosa bleeds slightly, the small quantity of coagulating blood adds to the size and often the color of the crusts. Saprophytic invasion may cause an offensive odor in the crusts.

Reflected Appearances—In the stage of *laryngitis sicca*, the mucosa is dull, dry, and rough in comparison with the smooth glistening appearance characteristic of the normal laryngeal mucosa. In the stage of atrophic laryngitis, crusts are seen adhering to the dry, patchy, eroded mucosa, in various locations, one or more may be seen partly detached. A common place for crusting is in or just below the posterior commissure, sometimes there are a number of small crusts on mucosal folds in this location. The color of the crusts varies, the crusts may be yellowish or grayish, or amber and brown when formed partly of blood.

Appearances on Direct Laryngoscopy—With the direct laryngoscope in place, an offensive, though not putrid odor is noted with each expiration. If crusts are seen one should be removed with a direct laryngoscopic forceps. It will be seen to leave a raw irritated surface that oozes a few tiny spots of blood. There is no ulcer, though part of the site from which the crust has been removed may show, in a spot or two, a slight superficial erosion of the epithelium. It is visible only by the oblique illumination afforded by the direct laryngoscope. The vertical rays of the mirror will not show it. The removed crusts usually have an offensive odor.

Diagnosis—The direct laryngoscopic appearances with removal of a crust, as described above, are diagnostic. The mirror may afford a working diagnosis. The atrophic, dull mucosa

of the larynx and the dryness of the crusts distinguish this disease from membranous tracheitis. Systemic syphilis should always be sought in these cases.¹

Prophylaxis—The arrest of the preceding chronic laryngitis is the only method of prevention. The methods are suggested under that heading.

Treatment—The mucosal glands being destroyed the only treatment is palliative. Residence in a warm moist seashore climate will greatly lessen crust formation, if humidity is very high (i.e., close to 100 per cent) no crusts at all are formed, the thick secretion can be expelled while pasty in consistency. Humidification of the home atmosphere is helpful, constant attention is necessary to maintain it. Sprays of liquid petrolatum inhaled two or three times daily may be used if humidification is impracticable, but otherwise they should not be used, as they lessen the penetration of water into the crusts. As a temporary expedient, normal salt solution may be injected into the larynx or inhaled as a spray.

Prognosis and Sequelae—The destroyed mucosal glands cannot be replaced, hence no cure is possible. The phonatory function usually remains impaired. There may be extension of the disease to the trachea, but even so the prognosis is not unfavorable as to duration of life, except in case of exceedingly dry and dusty environment.

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CHRONIC MEMBRANOUS LARYNGOTRACHEITIS

Chronic membranous laryngotracheitis is a condition of nondiphtheritic membrane formation without atrophy of mucosal glands. Fibrinous laryngotracheitis is a synonym. The disease is uncommon.

Etiology—In some cases the cause has been definitely determined as *Bacillus pyocyaneus*.¹ Fungi of various varieties have been causative. In two of our cases abundant aspergilli were present, the membrane being composed of a mat of mycelium.¹

Pathology—Grossly there seems only slight tissue change. The mucosa seems less inflammatory than might be expected. There is no ulceration, but oblique illumination from the distal lamp will show patches of superficial erosion of the epithelium. The membrane is made up of mucin, fibrin, leukocytes, epithelial cells, and granular material. In two of our cases it was chiefly matted mycelium.

Laryngeal Appearances—The membrane is usually seen projecting upward in the posterior commissure.¹ The supraglottic part of the larynx is rarely affected. *Tracheoscopy* and *bronchoscopy* show deeper membrane, if present. It may be patchy or continuous, and may affect only one bronchus.

Symptoms—Cough is somewhat annoying. Portions of membrane and sometimes casts are expelled. The casts may not be molded, but sometimes they show the form of the conus elasticus, the trachea, rarely the bronchi. Dyspnea during coughing attacks may be extreme when membrane is brought up from below the larynx. There is intermittent hoarseness, sometimes temporary aphonia. Hemoptysis is scanty, chiefly in form of streaks or small clots.

Diagnosis—The presence of clinging membrane in spots, patches, or as a detaching cast is diagnostic, so is coughing up of bits of membrane. In case of either of these features the absence of the bacillus of diphtheria after repeated examinations by culture is required for confirmation of the diagnosis. Diphtheria is not always an acute febrile disease but it never shows the extreme chronicity of this ailment. Any superficial traumatism may be covered with a grayish exudate but it disappears as the wound heals.

Treatment—In the cases due to definitely determined bacteria, as the *Bacillus pyocyaneus*, the disease may be cured by autogenous vaccines.¹ In the fungoid cases removal of membrane by direct endoscopic methods may be indicated when occlusion of the airway by a plug threatens asphyxia. A heavy spray of normal salt solution is helpful. Tracheotomy is not required when an endoscopist is available.

Prognosis—If asphyxia be prevented there is little danger to life. Ultimate return of all nine functions of the larynx is the rule, to which there are few exceptions. General health is always more or less impaired during the months or years the condition may continue.

Complications and Sequelae—The condition has few complications and no sequelae. Nodes may form in the neck, but usually do not suppurate, though they may remain palpable for a long time. Chronic membranous or fibrinous rhinitis may be associated, and the nasal membrane may contain *Corynebacterium diphtheriae* without systemic manifestations of diphtheria.

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CONTACT ULCER OF THE LARYNX

A contact ulcer of the larynx is a chronic ulcer located on the vocal process of one or more, often of both, vocal cords.¹ It is not uncommon but is often overlooked.

Etiology—The chief exciting cause is the hammering of one cartilaginous vocal process against the other, traumatizing the thin mucosal covering of both processes. It is really a form of indirect trauma. A perpetuating factor is a tiny area of necrosis of the cartilage underlying the tip of the vocal process. An important contributory factor is vocal abuse because of the incessant hammering. The disease usually occurs in incessant talkers or professional or occupational voice users. In the latter class must be included men who are required to abuse their voices by shouting to be heard above the noise of running machinery, for example. Dusty atmosphere and tobacco smoke may be added in such circumstances. The amount of abuse a larynx can stand is subject to a personal equation. It is found mostly in men but does occur, though rarely, in women.² It is almost never seen in children.^{1 2 3}

Pathology—A shallow ulcer involves the perichondrium in its bed. A tiny area of necrosis of cartilage is found in the bed of the ulcer in the cases of longer duration. In many cases a small mass of chronic granuloma covers the entire ulcer. The border of the ulcer shows a rim of inflammatory mucosa and submucosa, both of which are normally quite thin in this location. There may be a diffused chronic laryngitis but usually it is limited to the region of the ulcer. It is a curious and unexplained clinical fact that the ulceration and the tiny spot of perichondritis do not spread as in practically all similar lesions elsewhere in the larynx. It may be that the long time required for a break through the epithelium to be established by the hammering permits the building up of an efficient barrier in advance of the ulcerative process.

Reflected Appearances—In the mirror the ulcer is not conspicuous because it is presented edgewise. When large the prominence on one side is seen to fit into the cup of the other side on phonation, but both are often cupped.¹

Appearances on Direct Laryngoscopy—By tilting the top of the arytenoid eminence outward with the lip of the tube the bed of the ulcer is brought into view and in its center is seen a brownish rough spot, which is the necrotic tip of the vocal process of the arytenoid. The ulcer of one side, less often both, may be occupied by a granuloma of about the same color as the normal laryngeal mucosa. If superficially scalped off, the spot of necrotic cartilage is visible in the bed of the ulcer for a moment before it is obscured by a droplet of blood.

Symptoms—Hoarseness or, less often, only a slight impairment in the quality of the voice is noted except after prolonged talking. The voice gets worse and worse as the incessant talker keeps up his conversation. By evening the voice is usually weak and hoarse. It may be still hoarse in the morning on awakening, if so, it is likely to clear somewhat for an hour or two and then gradually grow worse again. Mild otalgia is not uncommon.

Diagnosis—A flat ulcer, its cup fitting over the prominence of the opposite ulcer with a spot of necrotic cartilage in the bed and at times a small granuloma, is diagnostic. There is no building up of pachydermatous tissue in the posterior commissure. In contact ulcer there is ulceration but no pachydermia; in pachydermia there is pachydermia but no ulceration. Biopsy

will confirm the diagnosis but taking of a specimen must be done with utmost precision in order not to include more than a tiny edge of normal tissue. The granuloma, if one is present, may be nipped off without any basal tissue and it alone will confirm the diagnosis, moreover, it may favorably affect healing processes in the ulcer. There is nothing to arouse a suspicion of cancer in the appearance of the ulcer, but any misgivings as to malignant developments could be eliminated by a carefully taken specimen for biopsy study.

Complications.—Strictly speaking there are no complications unless gross trauma is inflicted surgically or by ill advised cauterization. Contact ulcer, however, may be concurrent with other diseases such as systemic syphilis or pulmonary tuberculosis, for example.

Treatment.—The regimen of silence as given in consideration of treatment of myasthenia laryngis is the basic treatment for contact ulcer also. Granuloma should be nipped off superficially as advised for vocal nodules (q v). Nipping out the ulcer, or nipping off the necrotic tip of cartilage, will not hasten cure much, if at all, because of recurrence. Nitrate of silver and similar applications are worse than useless. The galvanocautery is likewise objectionable. Elimination of vocal abuse, including change of occupation if necessary, is the essential thing.

Prognosis.—There is no danger to life and, if no injudicious surgery be done, all nine functions of the larynx will be restored by the regimen of silence, if the patient will cooperate perfectly. The course of healing is slow, the patience of the patient and the family, even of the family physician, may become exhausted. Recurrence may follow relapse into vocal abuse. There is no danger to life.

Sequelae.—There are none if the condition receives attention without too long delay. If allowed to continue until a mass of fibrosis is built up there will be permanent impairment of the voice.

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MYASTHENIA LARYNGIS

Myasthenia laryngis is defined as disability of the pharyngeal muscles of the larynx. It has no relation to myasthenia gravis. It is a common ailment among persons who use the voice occupationally. Accurate statistical data are unobtainable because most of the cases are recorded as chronic laryngitis which is a mucosal inflammation that may or may not coexist.

Etiology.—The exciting cause is indirect trauma in the form of overwork of the laryngeal muscles especially the thyroarytenoid. This is always in the form of vocal abuse, though other factors may contribute to the abuse by throwing an extra load on the muscles—a growl between the cords or a stiffened cricopharyngeal joint, to cite two common examples. Both of these conditions inevitably require increased muscular exertion in the effort of the victim to force a better tone. Another example is the effort to be heard above the noise of our needlessly noisy civilization. Countless thousands of our people run their voices by forcing their respective larynges to produce a tone loud enough to be heard above unnecessary noises. The associations for the abatement of noises might well add this to their propaganda for reform. The indirect trauma may be occupational as in persons who must talk in noisy places, for example where noisy machinery is running, or riveting is being done. Continuously talking to persons with defective hearing is causative in some cases. One of the largest groups of cases includes professional voice users. These persons are in every degree of the social scale—teachers, hucksters, train announcers, auctioneers, college professors, stump speakers, politicians, statesmen, ministers, clergymen, evangelists. The largest single group is composed of singers.

Of those who start out with an ambition for a great career in vocal music probably more than 95 per cent never attain it, and most of the promising vocalists who fail do so because of myasthenia laryngis. The laryngeal

muscles, especially the thyro-arytaenoid, cannot stand the strain of the years and years of grueling daily practice necessary to reach the top in operatic and concert solo work. Few persons realize that the possession of a good larynx does not make a great singer any more than the possession of a genuine Stradivarius makes a great violinist. In either case no talent will obviate the necessity for a lifetime of daily hours of practice. To make matters worse most singers talk all the time they are not singing in performance or practice. The laryngeal muscles get no rest. Additionally, all sopranos and tenors are under incessant pressure all their professional lives to increase their vocal range upward in pitch. The strain on the thyro-arytaenoid is extremely severe and in most instances ruinous. Faulty methods as all vocal teachers know, are sometimes partly to blame, but few human larynges can stand the strain of constant severe work and no rest. Another causative factor is forced use of the larynx in performance and practice while the larynx is acutely inflamed. All singers feel three compelling impulses to perform during an acute laryngitis: first, there is the natural feeling of reluctance to disappoint the audience, second, there is the compelling tradition among all public performers that 'the show must go on', third, nearly all great singing voices are ultimately 'lost,' next to dread of losing the voice is the dread that someone will start a damaging rumor that a singer is losing his voice. All of these factors result in forcing every great singer to sing during an acute laryngitis such as everyone gets occasionally. This is one of the worst forms of injury anyone can inflict on his larynx using it during an acute laryngitis (q.v.). More over, to prepare for performance, there is the additional injury resulting from the necessary rehearsals, in these and in the performance more than usual force is necessary to get a satisfactory tone out of the inflamed larynx.

The fundamental principles of causation of myasthenia laryngis in singers are the same as in the cruder forms of professional use of the voice, except that in the other professions the same amount of rehearsal is not required nor is the quality of the voice the cynosure of all critical attention. Yet a professional public speaker's voice is a great asset and he risks its ruin by myasthenia laryngis due to vocal abuse.

Vehement exhortation may cause myasthenia laryngis by the infliction of sudden, violent indirect trauma. The same is true of such vocal abuse as screaming and shouting, also the cheering at baseball and football games. The reckless enthusiasm engendered by alcohol may add to the ruinous force in the use of the laryngeal muscles.

Pathology.—The pathologic condition in myasthenia laryngis is a muscular disability. It may be in the form of muscular injury due to sudden violent use, such as the strain that commonly occurs in the skeletal muscles, but oftener it is due to the strain of prolonged as

well as violent overwork without proper intervals of rest. Work of the laryngeal muscles within proper limits increases their strength, but overstraining and overwork produce a disability from which the muscles do not recover. 1, 2

Reflected Appearances.—To get a proper appreciation of the appearances in myasthenia laryngis it is necessary to be thoroughly experienced in the images of the normal, especially of the normal phonation in singers (q.v.). The chief characteristic of the appearances in most cases of myasthenia laryngis is failure to hold the set of the larynx for tone. The muscles of the geying group usually act promptly. The orbicular group follows, sometimes promptly and at other times sluggishly. In either case the perfect set is not held for a long time. The glottis is seen gradually to lose its shape, the edges separate, and the tone loses its clarity and usually drops in pitch. In some cases the tone is suddenly lost, and the chunk of the whole membranous glottis becomes elliptic, with flaccid margins, denoting complete failure of the thyro-arytaenoid, followed by relaxation of the cricothyroid. In many cases the typical appearances just described are not manifest at the first in spection, and they may or may not appear until the patient has made many phonations with the mirror in place. In still other cases the myasthenic appearances can be found only by having the patient come in after fatiguing practice or performance. In these cases the disability is in an earlier stage, and the patient sometimes gets through a performance fairly well. Examination is usually best made when the voice has "broken," "cracked," or "let down." In all such cases it is important to make, and record for comparison, a mirror examination at an interval when the voice is at its best. This is usually after a period of vocal rest, but it is well simply to ask the patient to come in when the voice seems most responsive to control. The typical appearances described may be altered in some cases by spasmodic action of the orbicular group of muscles, but rarely by the geying group. When the patient endeavors to phonate, there is a snapping shut of the glottic chunk, quickly followed by relaxation of the glottic margins into a more or less elliptic form. Sometimes this picture is noted before the patient attempts to phonate. The mucosa may be normal, though usually it shows the vascular engorgement secondary to forceful phonation. Due allowance must be made for

the fact that the mucosa of singers (and of professional voice-users as a rule) is more vascular than that of the average person who does not use the voice excessively

In differential diagnosis, diseases of the recurrent laryngeal nerves are to be excluded. All organic lesions, such as tumors in the conus elasticus impairing movement of the lateralis or of the thyro arytenoid muscle, must be excluded, and the same may be said as to the other muscles concerned in phonation. The possibility of myositis, arthritis, and disease of the laryngeal cartilages can be eliminated by a thorough laryngeal examination in all of its four phases.¹ In cases of occupational compensation claims the laryngologist will be able to detect malingerers by the evident lack of best effort at phonation.

Prophylaxis.—The prevention of myasthenia laryngis in professional and occupational abusers of the voice, as well as in incessant conversationalists, is easy in principle but difficult in practice. It is useless to tell these patients that they should rest the larynx. They must be given specific orders. The following *regimen of vocal rest* has stood the test of years of clinical work with laryngeal disease.

(1) A period of absolute silence is necessary every day, in addition to hours of sleep. (2) The silent period should be at least half the duration of the strenuous occupational period. (3) On days of especially strenuous vocal work all nonoccupational use of the voice should be cut out, this, including hours of sleep, would give sixteen hours of vocal rest daily. (4) Absolute silence should be maintained over the week end. (5) All strenuous use of the voice such as talking against a noise, for example in a subway train or in the street, should be avoided. (6) Any necessary use of the telephone should be in a low tone with the telephone transmitter held close to the lips. (7) It must be remembered that, although strenuous vocal work is most severe on the larynx, talking, humming and even whispering call for work by the laryngeal muscles that rob them of needed rest. (8) In case of singers a special rule should be added to the foregoing. Striving to increase the range of pitch upward is a great strain on the muscles of the larynx. It must be done in moderation and calls for counterbalance by a long daily period of absolute silence not less than eighteen hours, including those of sleep, out of the twenty four. It would seem better for the young vocalist, in many instances, to be contented with a good and permanently good, voice rather than to risk losing the best quality of voice by striving to extend the range upward beyond safe limits.

Treatment.—Local applications are useless in the treatment of the overworked and disabled laryngeal muscles. The treatment re-

quired is the *regimen of silence*, of which the following is an outline to be modified according to circumstances of the particular case.

(1) A preliminary period of silence with a total allowance of say, 100 words a day spoken in a low tone, of natural pitch, in quiet places is essential. The duration of this preliminary period of silence should be from one to six months according to the severity of the case. (2) After the preliminary silent period the use of the conversational voice is resumed gradually over a period of a month or two, but only in quiet environment. (3) The next period is the gradual resumption of the professional or occupational use of the voice. This use must be counterbalanced by eliminating all conversation. (4) The patient is put upon the regimen of vocal rest outlined in the preceding paragraph for many months to overcome his bad habit and to replace it by a habit of always thinking of the necessity for keeping within reasonable limits before he speaks. (5) In the case of singers, after the preliminary rest period the patient may be allowed to start all over again in vocal training, keeping strictly within the natural range and avoiding all attempts at extension of pitch upward. Only after a year or so of this limited training may the patient be allowed to commence extension upwards and this should be only by slow stages. At the first sign of return of the trouble efforts at extension must be dropped for months, and the regimen of vocal rest mentioned under 'Prophylaxis' must again be followed.

One of the pitfalls in treatment of myasthenia laryngis in singers is the supposition that some different system of vocal training or some special exercises will cure damaged thyro-arytenoid muscles. This is a fallacy. No training or special exercises can be carried out without working the already jaded muscles. It is true that the training that brought on the trouble may have been faulty or even ruinous, but before a better system is undertaken a regimen of silence, as outlined, is necessary to allow the muscles to recover from the damage previously done. The best advice to give the great singer with impending myasthenia laryngis is to retire before disastrous failure causes lifelong regret.

Prognosis.—When the condition is recognized early and the patient cooperates perfectly in maintenance of the regimen of silence, the prognosis is good. If the patient insists upon constantly talking during intervals between professional work periods the prognosis is bad as to voice. In a case of advanced myasthenia laryngis in a great singer in the later years of a great career, the prognosis as to continuance is hopeless, and the singer should be so advised. If the patient, after a regimen of rest, will be contented with a little humming and an occasional bit of song in a small room for a few

friends a satisfactory singing and conversational tone may be retained, but the concert and opera are to be regarded as things of the past in such cases. Public speakers ordinarily may count on a moderate amount of speaking with long intervals of rest. Voice as such is not under criticism so much as what they have to say. To the singer the alternative to perfect tone is utter failure of the musical career. Train callers and similar abusers of the voice, if the myasthenia is well advanced, rate a total permanent disability for that particular occupation, though they can, with care, retain a good useful voice for relatively silent occupations. Obviously there is no danger to life in myasthenia laryngis.

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PERICHONDRITIS OF THE LARYNX

Inflammation of the perichondrium that covers the laryngeal cartilages is known as "perichondritis of the larynx." It is still a relatively common laryngeal disease. Though less frequent as a complication of general disease than in the days of less efficient control of diphtheria, typhoid fever, tuberculosis, and syphilis, there has been a progressive increase in traumatic cases in recent years.^{1 2} There was a largely increased incidence in World War I and it is still larger in World War II.

Etiology.—Infection is one causative factor in practically all cases. The infective agent, the atrium, and the manner of access vary widely. Direct infection usually occurs in most of the many forms of trauma (*qv*). The infection may reach the perichondrium indirectly in the course of chronic diseases such as syphilis, tuberculosis, and cancer. Among acute diseases complicated by perichondritis the most common are typhoid fever, diphtheria, variola, measles, influenza, pneumonia, scarlet fever,

erysipelas, blood dyscrasias, typhus, tonsillitis, peritonsillar abscess, Vincent's infection, Ludwig's angina, anthrax, and mycoses.^{1 3} Irradiation was a common cause before radiologists realized the exquisite sensitivity of the laryngeal cartilages to both radium and the roentgen ray.⁴ Of the causative bacteria, hemolytic streptococci are most frequently found predominating, but all the other common pyogenic organisms are found, sometimes in nearly pure culture. Even in the specific infections, such as diphtheria, typhoid fever, syphilis, and tuberculosis, the structural damage is usually done by the secondary invaders.^{1 3 5} Oral sepsis is a contributory factor.³ Tracheotomy too close to the larynx and ill-advised after-care of the tracheotomic wound are common causes of laryngeal perichondritis.² Underlying all causative factors is the fundamental anatomic fact of the poor reparative power of cartilage.

Pathology.—The dense connective tissue composing the external fibrous layer of the perichondrium is a strong barrier against infection, but once infection gets through that barrier, directly as in trauma, or by continuity, or by way of the vessels perforating the fibrous layer, the vulnerable chondrogenetic layer is reached. Destruction of this layer quickly follows, and with it goes the dependent nutrition and power of repair of the cartilage. Dead cartilage becomes a septic foreign body in an infected suppurating focus. Even if drainage is established, spontaneously or surgically, lack of nutrition of the cartilage, because of extending destruction of the chondrogenetic layer of the perichondrium, results in progressive extension of chondral necrosis. Thus we see in typhoid fever, for example, extrusion of the arytenoid cartilages, each as a sequestral unit.³ Loss of cartilaginous framework to resist contraction of the fibrous tissue built up during the long healing process is a large factor in cicatricial stenosis (*qv*).¹ Owing to the calcification and ossification of the laryngeal cartilages after about the twentieth year in men, thirtieth in women, chondral necrosis in some cases takes on the character of an osteomyelitis.¹

Reflected Appearances.—Swelling is the general characteristic of perichondritis when the larynx is examined with the mirror. The location of the swelling depends upon the site of origin and the stage of the disease. If one arytenoid is first affected it will be swollen, smooth,

and rounded out of proportion to the other arytenoid. Except in sluggish diseases, like tuberculosis, perichondritis does not long remain thus limited. In typhoid fever both arytenoids are usually seen to be about equally swollen. When the inner perichondrium of the thyroid cartilage is affected in any type of inflammation a bulging inward of the affected side is noted early as a localized formation, but later the entire wall is seen mounded inward with landmarks obliterated. At a later stage, when the perichondritis has become diffused, there are usually three mounds, one anteriorly representing the epiglottis and one on each side representing the arytenoid eminences and ary-epiglottic folds, with landmarks obliterated. The color of the swollen tissue varies. It is usually red in the early stages of acute diseases, and the redness is localized at the primary focus. As the process spreads the redness is diffused over part or all of the larynx. Soon, however, the reddish color becomes paler because of edema and may get almost white. When perichondritis develops in typhoid fever, in advanced tuberculosis (pulmonary and laryngeal), and in other anemic conditions, the endolaryngeal swellings are pale from their beginning and progress is sluggish. In most other diseases the appearances at a relatively early stage are those of edematous swellings obliterating the lumen so that the interior of the larynx cannot be seen in the mirror.

Appearances on Direct Examination.—By gentle use of the laryngoscopic tube mouth the interior of the larynx may be explored and a good view of parts concealed in the reflected image, by the swollen and edematous tissues, can be obtained. Moreover, the tissues can be instrumentally palpated, and exogenous foreign body may be found or excluded from among diagnostic possibilities, the same may be said of an endogenous foreign body, such as a sequestrum of cartilage. In a number of such cases we have found the sequestrum to consist of ossific material and to be of foul odor. If a purulent accumulation has been spontaneously evacuated a fistulous orifice, with flabby granulations, may be found. Gentle probing may convey the dull rubbing sensation of cartilage or a gritty sensation will be felt if there is denuded calcareous or ossific cartilage. If pus underlies the bulging tissues at any point a boggy sensation will be transmitted to the closed forceps used gently as a probe.

Symptoms.—Hoarseness, a "thick" voice, dyspnea, dull pain (aching in character), slight cough (usually productive), and pain or soreness on swallowing are the chief symptoms. Slight fever may be present in acute cases, but may be attributable to the basic malady.

Diagnosis.—The reflective endolaryngeal appearances are suggestive, the direct appearances previously described are diagnostic. *Palpation* (*q v*) is an absolutely certain means of diagnosis. Tenderness on pressure is present whether the inner or the outer perichondrium is affected, if the outer, the finger will quickly locate the spot of tenderness and swelling under the perichondrium. Serologic tests for syphilis and blood examination for dyscrasia should be done in every case, regardless of other findings. Roentgen-ray examination may be helpful in locating foreign bodies or purulent collections.

Complications.—Necrosis of cartilage complicates almost all cases. Abscesses, endolaryngeal or cervical, are common complications.

Varieties of the Disease.—Perichondritis may be acute or chronic. For example, it is acute in diphtheria and variola, chronic in tuberculosis.

Prophylaxis.—Efficient oral hygiene is important as a preventive,³ so are early diagnosis and efficient treatment in such diseases as diphtheria, the mycoses, the blood dyscrasias, and syphilis. Specific drugs (*q v*), by arrest of infective inflammation of adjacent tissues before it extends to the perichondrium, are powerful prophylactic agents. The sulfonamides and penicillin have been successfully used for prophylaxis.

Treatment.—A close and constant watch for the cardinal signs of obstructive laryngeal dyspnea (*q v*) should be maintained and preparations for tracheotomy should be made in advance. The opening should be made low into the trachea. Intubation may be preferred for diphtheria, even so, preparations for tracheotomy should be made, anyway. Systemic treatment of the basic disease is of primary importance for the arrest of the perichondritis, especially in such diseases as diphtheria, syphilis, and the blood dyscrasias. In such cases, local treatment should be limited to prevention of asphyxia. In all pyogenic infections, prompt use of specific drugs (*q v*) is of utmost importance. In all cases, even those of the most chronic type, local treatment should be conservative. Pus may be evacuated by incision with the knife (Fig. 350) under precise guidance of the eye, by means of

the direct laryngoscope. With laryngeal forceps it is easy to remove a foreign body. In case of a sequestrum, it may be removed if completely detached, but it should not be pulled upon if not detached. Fistulous orifices are to be kept open by biweekly dilatation using the alligator jaws of the laryngeal forceps (Fig. 341) expandingly. If pus be felt under the skin externally it may be evacuated through a conservative incision. If the perichondritis is felt to be still in the stage of swelling the possibility or even probability of breaking down into pus does not warrant following the rule of surgery to "lay it freely open." It should be opened with precision and when pus begins to form, not when only swelling is palpable to the trained finger. It must be remembered, regarding the epiglottis, the thyroid, cricoid, and the two arytenoid cartilages, that each is isolated in its own perichondrium. It is a disaster in liberating pus from one cartilage to puncture or incise, even minutely, the perichondrium of an uninvolved cartilage. A new focus of perichondritis and chondral necrosis will surely follow. Each cartilage is an integral part of the laryngeal framework, without which a normal lumen cannot be maintained. External incisions must be packed open until extrusion of sequestra and fibrous union are complete. Cartilaginous union is not to be expected in adults and is rare even in children. As soon as chondral necrosis has ceased to progress, core-mold treatment (*q. 1*) should be commenced to avert stenosis of the laryngeal lumen, or gradually to enlarge it. It is of utmost importance to prevent total atresia, even the most slender core mold will accomplish this.

Prognosis.—Of all laryngeal diseases and complications, perichondritis carries the greatest threat to the laryngeal framework and most of the nine functions of the larynx. If asphyxia be prevented, laryngeal perichondritis has a low mortality rate considered apart from the illness of which it is a complication, not over about 3 per cent, in our experience. As a phase of the late and terminal stages of typhoid fever, tuberculosis, and cancer, for example, it would take on the hopeless prognosis of these conditions. If the mortality is calculated inclusive of the patients who die for want of a tracheotomy about 11 per cent would be added to the 3 per cent above mentioned.

Sequelae.—Stenosis and total atresia of the larynx were formerly the end result in almost

all cases. Early application of modern methods of prophylaxis and treatment have placed both of these conditions in the potentially preventable or the curable class.

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ACUTE OBSTRUCTION OF THE LARYNX

Acute obstruction of the larynx may be defined as a laryngeal obstruction of sudden onset or rapid progress. The distinction of suddenness is important clinically. *Edematous laryngitis* is a loosely applied term often used when the obstruction is not an edema and not even inflammatory. Acute laryngeal obstruction is not uncommon.

Etiology and Pathology.—Trauma, foreign body, and disease are the three classes of the many causes of acute laryngeal stenosis. These are considered under their respective headings, but it will save space and be conducive to clarity if the clinical features and proper treatment generally applicable be separately stated here. Morbid obstruction may be *noninflammatory* (as in angioneurotic edema and bilateral laryngeal paralysis), *inflammatory* (as in peri-

chondritis), or *neoplastic* (as in cancer) Whatever the primary cause may be, pyogenic bacteria are secondary invaders if the epithelial barrier is broken Hemolytic streptococci are the most frequently found but are not the only secondary invaders Autopsy, in cases of asphyxia from laryngeal obstruction, may show pulmonary atelectasis without much laryngeal stenosis because of conditions explained under "Symptoms", in cases of midline abductor laryngeal paralysis the larynx may look quite normal at autopsy

Laryngeal Appearances—These vary with the pathologic conditions present but the important point to determine is the apparent area of cross section of the glottis at its narrowest point, and this determination must be made *on inspiration* It cannot be too strongly emphasized that whereas the greatest possible area of cross section of the normal laryngeal airway is on deep inspiration the exact contrary is true in practically all obstructive morbid conditions

Symptoms—The chief symptom is *obstructive laryngeal dyspnea* The cardinal signs of this condition are indrawing on inspiration, pallor, and, in children, restlessness The indrawing on inspiration is most, and first, noticeable at the suprasternal fossa, less and later noticeable around the clavicles, in the intercostal spaces, and, in children, at the epigastrium (funnel breast) The pallor is sometimes dark gray or bluish, or violaceous, but in seriously ill children it is ashy gray *Restlessness* due to air hunger is one of the most important early signs, not only because it is significant but because if misunderstood fatal sedatives may be given to overcome it It cannot be too emphatically stated, however, that the restlessness ends in fatigue and that a child tired out and oxyc will sleep without a struggle for air Every time the child falls asleep he loses the aid of the voluntary muscles of respiration Eventually need for sleep becomes overpowering and the child sleeps quietly as respiration ceases

Laryngeal dyspnea is practically always inspiratory for a number of mechanical reasons—one of which is shown in Figure 357—all of which are fundamentally due to the fact that for millions of years, dating from its earliest evolutionary stages, the chief function of the larynx was protective (see under "Physiology of the Larynx") Stridor is usually present and it is inspiratory also The character of the sound is usually more or less fluttering, but may be

relatively smooth and sonorous as in the whoop of pertussis The severity of the symptoms, especially the inspiratory dyspnea, is dependent on the rapidity of onset In cases of chronic stenosis in which an extreme degree of stenosis has slowly and progressively increased during a number of months, the patient has learned that the more violent his efforts to inspire air the less air he gets, and has learned to inspire slowly and quietly with least possible exertion

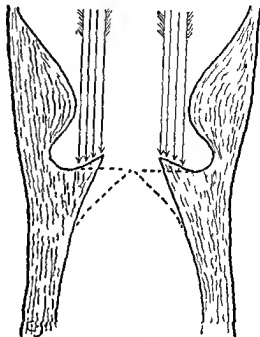


Fig 357—Simplified illustration of the fundamental anatomic fact explanatory of the clinical fact that laryngeal dyspnea is practically always inspiratory Because of the slant of the ventricular floors the inspiratory inrush of air, indicated by the arrows, slams the glottis shut like a pair of hinged cellar doors The more sudden and violent the inspiratory inrush the quicker and tighter the closure Slow, gentle inspiration makes little pressure on the ventricular floors Expiratory air-current upward through the conus elasticus blows the glottis open

When obstructive laryngeal dyspnea is sudden in its onset the patient, frightened by the sensation of suffocation, makes violent inspiratory efforts that close his laryngeal airway He may even thus asphyxiate himself, although he has as much airway as the chronic patient who gets along comfortably because he has learned the advantage of effortless gentle inspiration (Fig 357)

Diagnosis.—The diagnosis of the character of the obstruction is made by endolaryngeal examination with the mirror or the direct laryngoscope, the latter being the only practicable

method in younger children. The diagnosis of the presence of obstruction does not depend on laryngeal examination; it is based on the previously described objective signs of obstructive laryngeal dyspnea and these are obvious from the other side of the room.

Prophylaxis—The disease may or may not be preventable, but death by asphyxia can be prevented if the patient comes under surgical care before the heart has ceased to beat. Usually it ceases within three or four minutes after cessation of respiration.

Treatment—A foreign body exogenous or endogenous can be removed by direct laryngoscopy. Obstructive secretions and exudates can be removed by direct laryngoscopic aspiration (Figs. 469-470). Benign tumors can be removed by direct laryngoscopy (qv); malignant tumors by external operation. Intubation may be used in diphtheria if an expert intubator is to be constantly available for reintubation in case the tube should be spontaneously expelled. For all other conditions early and low tracheotomy is indicated in almost all cases. If the cardinal signs are not urgent, tracheotomy may be postponed for a time if a tracheotomist is promptly available, but as a rule when the signs mentioned are present in the slightest degree the patient should not have an unwatched moment. If the patient is an older child or an adult and is not unconscious, first aid consists in calmly convincing him that he can get more air and great relief if he does not try so hard to pull in air. But not a moment should be lost in doing a tracheotomy (qv). The treatment of the various morbid conditions producing obstruction is considered under the heading of the respective conditions. The inhalation of oxygen is excellent, being based on the fact that it compensates for the diminished air supply by increasing the percentage of oxygen in the inspired air. If the patient has ceased to breathe, artificial respiration is necessary to draw in the oxygenated air. Best of all means of relief of extreme dyspnea and resuscitation after breathing has ceased is bronchoscopic oxygen insufflation. As explained in a subsequent paragraph when oxygen is used for dyspneic conditions it should always contain an admixture of about 7 per cent carbon dioxide.

Prognosis—Almost all of the conditions causing laryngeal obstruction are curable. Considered apart from any systemic disease that may coexist and apart from the deaths for

lack of a timely tracheotomy, the prognosis is good. In children, prolonged deep anoxia may damage respiratory centers to such a degree that recovery becomes impossible.

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ASPHYXIA

Death caused by lack of oxygen in the tissues is said to be due to asphyxia. About 14,000 deaths are annually recorded as asphyxial in the registration area of the United States.¹ Statistics, however, do not afford even an approximate idea of the number of deaths due to asphyxia. Lack of oxygen in the tissues is the terminal and decisive phase of death from most of the diseases that afflict mankind, for example, it is a common occurrence for the heart to give out in pneumonia for want of oxygen in the heart muscle, yet such a death is never recorded as a death by asphyxia. Practically all deaths recorded as due to drowning are asphyxial deaths; the same is true of deaths by carbon monoxide poisoning and electric shock. From these and other direct causes an enormous number of asphyxial deaths occur every year, and it may be added that a large proportion of them may be classed as preventable. Every physician occasionally encounters cases of impending asphyxia—the laryngologist frequently the obstetrician, often the bronchoscopist almost daily.

Etiology—The most frequent cause of unrecorded asphyxia is respiratory failure in death of one of the slower types. The heart stops for want of oxygen in its muscles. A com-

mon unrecorded cause is foreign bodies, especially endogenous, of which the number of cases is enormous, these foreign bodies include plugs of mucus, exudates, crusts, membranes, inflammatory products, and blood. Occlusion of the smaller air passages by swelling of the walls occurs in all pulmonary diseases. Of recorded or recognized types of asphyxia the most frequent cause is failure to establish the respiratory function in newborn infants. Many of these infants are recorded as stillborn. Other common causes are drowning, electric shock, inhalation of carbon monoxide and other lethal gases, and trauma (thoracic, tracheal, laryngeal, or central). A detailed list would include hundreds of causes. The cause of asphyxia of babies found dead in cribs is often too many or too heavy covers too tightly tucked in. If a single light-weight coverlet were spread over the baby and not tucked in he could easily turn over and free himself when his mouth gets covered deep in the pad, mattress, or pillow, if one is used.

Pathology—The fundamental condition is lack of oxygen in the tissues (anoxia). Another fundamental fact is that this anoxia soon results in a deficiency of carbon dioxide in the blood (acapnia), because oxygen is an essential raw material from which the system normally manufactures its vitally necessary supply of carbon dioxide. Crudely expressed, oxygen is a food that cannot be assimilated without the co-operative stimulation of carbon dioxide in the blood. In impending asphyxia, both oxygen and carbon dioxide are deficient and both must be supplied if we are to avert an asphyxial death. These are the fundamental facts we need to know for practical work, the correlated physiologic processes are among the most complex and interesting problems in medical science.¹

Symptoms.—The vitally important prodromal symptoms of impending asphyxia due to laryngeal obstruction are those of obstructive laryngeal dyspnea (p. 473). As respiration fails it becomes slower, then convulsive and gasping just before it ceases. After the respiratory murmur disappears the last few respirations produce an abundance of mucus râles in the bronchi. The heart becomes irregular, then slower and ceases in diastole. The pupils dilate widely, fail to react to light, and all other reflexes are absent. The color of the skin is usually violaceous, except in exsanguinated victims, in whom the color may be bluish white.

Diagnosis—The symptoms are diagnostically unmistakable to anyone who has ever seen a patient with impending asphyxia. There is a momentary similarity between the livid face of an epileptic who falls unconscious in a major attack and the livid face of a man who falls suddenly suffocating from occlusion of the larynx by a bolus of meat, for example. Though both are cyanotic, air comes frothy through the clenched teeth of the epileptic, his convulsions are greatest while he is unconscious, his color improves and his breathing becomes regular as the convulsions cease. The suffocating victim is making violent respiratory movements but no air is passing through his widely open mouth, when his struggles cease his color becomes more intensely vivid and he is limp. The eyelids of the epileptic convulsively snap open and shut, whereas the victim of impending asphyxia stares wildly with widely open eyes. The only way to make a diagnosis among the many laryngeal obstructive conditions that cause asphyxia is to look at the larynx. If an adult patient is approaching unconsciousness the direct method of examination should be used. In children it is the only possible means (see under "Asphyxia Neonatorum," p. 477).

Prophylaxis—Inasmuch as the word "asphyxia" means death the only treatment to be considered is prophylactic in character. The fundamentals are these: (1) clearing of the airway, (2) getting oxygen into the tissues, and (3) stimulating the respiratory center.

For clearing the airway nothing equals, in efficiency, direct laryngoscopy and removal of obstructive material with the aspirating tube and, if necessary, forceps, under guidance of the eye. The ordinary aspirator used in tonsillectomy will clear the pharynx but cannot be used by sight in the larynx. If no instrumental equipment is available the laryngopharynx and mouth may be wiped out with a handkerchief. The day is not distant, however, when it will be considered deplorable, if not criminal negligence, not to have available everywhere the simple equipment required, and someone trained in its use, as indicated in Figures 469, 470, and 471 (pp. 628, 629).

There is only one route by which oxygen may be gotten into the tissues—namely, the blood stream, and there is only one way, as yet known to medical science, by which oxygen can be gotten into the blood stream, and that is through the walls of the air vesicles of the lungs. As air

contains only about 20 per cent of oxygen the oxygen air content should be increased to compensate for diminution of air supply. The simplest and most efficient way to accomplish this is by direct laryngoscopic insufflation or bronchoscopic insufflation of oxygen (Fig 358). In the absence of tanked oxygen the best atmospheric air available must be caused to circulate in maximum possible quantity through the lung by artificial respiration.

The best of all *respiratory stimulants* is the natural one carbon dioxide. The simplest and best way to administer it is as a 7 per cent ad-

places the clearing of the tracheobronchial tree and the insufflation of oxygen and carbon dioxide under perfect control.

Asphyxia in the Operating Room—For emergencies nothing equals intratracheal insufflation of oxygen with 7 per cent carbon dioxide admixture. Every anesthetist and every interne should be trained in the simple technique required (Figs 469, 470, 471). The instrumental outfit is simple and inexpensive. Respiratory movements are not absolutely necessary to ventilate the lungs; insufflation delivered near the tracheal bifurcation is really sufficient, but artificial

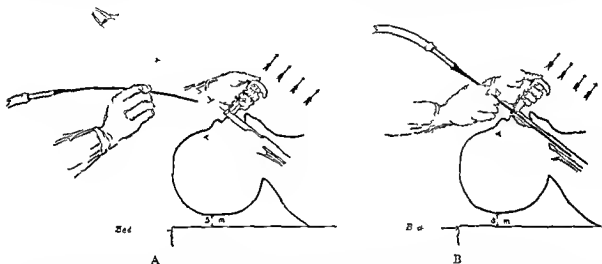


Fig 358.—Simplified schematic sketches illustrating direct laryngoscopic aspiration and oxygen-carbon dioxide insufflation in presence of asphyxia neonatorum. A Sketch shows the technique of exposure of the larynx by the parallel lifting of the head forward as indicated by the dashed arrows. The distal end of the laryngoscope covers about 6 mm of the tip of the epiglottis. B When the orifice of the larynx is seen just beyond the epiglottis, the silk woven aspiration or insufflation tube (No. 8 F, never larger in the newborn) is inserted into the trachea, not deeper than 2 cm. After aspiration a gentle flow of oxygen-carbon dioxide (7 per cent) mixture is passed through the tube. No effort is made to inflate the lung by positive pressure, to do so would be fatal. All manipulations must be gentle. Precisely the same method is used in children of any age for dyspnea or other obstructive conditions, but the silk woven tube may be larger in older children (Figs 469, 470, 471).

mixture to the oxygen.² Tanks with this admixture are now commercially available everywhere and should be constantly on hand at all hospital accident wards, police stations, fire houses, first aid stations, ambulances, patrol cars, seashore life saving stations, drugstores, and other locations where they would be quickly available. Lack of such availability is responsible for thousands of asphyxial deaths every year.

All of the three fundamental requirements for the prophylaxis of asphyxia are best dealt with by emergency tracheotomy (q.v.) and ordinarily the sooner it is done the better. It can be performed in from one to two minutes and it

respiration should be added if all respiratory movement has ceased. With a patient recumbent under operation, Silvester's method of artificial respiration is the best and it has the additional advantage that it can be carried on without interfering with the insufflation. Caution is necessary to avoid any ballooning of the lung by forced pressure. Alveolar walls are easily ruptured, but far short of this is the slight increase of pressure necessary to compress the capillaries and force the blood out of the alveolar walls.²

Nonmedical First Aid—All the foregoing methods applicable to impending asphyxia require medical skill. Unfortunately a large pro-

portion of these cases occur where no physician is available. In such cases what is to be done in a particular case depends upon the persons and materials available at the time and place. The excellent *prone pressure method of artificial respiration* is now taught everywhere, to Red Cross workers, police, firemen, nurses, boy and girl scouts, even to high school pupils. The mouth should be low enough to let secretions escape. If low enough the tongue may fall forward enough to clear the backwall, but for certainty the tongue should be held forward by any available helper to eliminate the *lingual death zone*.^{1 2} If the tongue is not kept forward it operates as a one-way valve, permitting egress of air at each pressure stroke, but plugging the laryngeal orifice against admission of air at each inspiratory phase, in other words, pumping the lungs empty. This fact should be added to the teaching of the excellent prone pressure method.

Asphyxia Neonatorum.—This term is applied to cases in which neonatal respiration is not established. It does not apply to cases of congenital laryngeal stridor (*qv*). The highest human death rate occurs in the first fifteen minutes after birth. The mortality rate in this short period is over two thousand times greater than at any other period of life. Great as it seems this mortality is small compared to what it was prior to the modern development of obstetric science. Asphyxial deaths constitute a large proportion of the high neonatal mortality, and in a large proportion of these cases death is due to obstruction of the air passages. Obviously, prevention of asphyxia is hopeless unless the obstruction is removed. It has long been the obstetric routine to clear from the nose and mouth the secretions incidental to childbirth, but this clearing of the upper air passages has been supplemented by the clearing of the deeper air passages with the development of direct laryngoscopic inspection and aspiration (Fig. 358). This supplementary clearance of obstruction by direct means carries with it the insufflation of oxygen and its 7 per cent complement of carbon dioxide, which prevents asphyxia until respiratory movements are established. Obstruction in the larynx, trachea, and larger bronchi is usually by inspiration of mucus, vernix caseosa, maternal secretions, amniotic fluid. Unquestionably an occasional respiratory movement occurs in the late stage of intra-uterine life as well as

in prolonged labors, especially when associated with malpresentation. In such cases the protective function of the larynx has not yet been established. Moreover, there is yet no air below the larynx with which to create the equivalent of a bechic blast. The only natural spontaneous means of clearing the neonatal airway is the compression of the lungs by the thoracic muscles, in a way mechanically similar to what later becomes the tussive squeeze (*qv*). If these muscles are inadequate for the task the infant asphyxiates. The infant laryngoscope (*qv*) enables the obstetrician of today to inspect the laryngopharynx and the larynx, and to clear the laryngotracheal airway by aspiration (Fig. 358). Gentle insufflation of oxygen-carbon dioxide mixture can then be carried on until the child can be trusted to do his own breathing, after which the tube can be withdrawn.² It seems necessary to repeat here for emphasis that there must be no thought of inflating the infant's lung by positive pressure, this, probably, would be promptly fatal. As elsewhere herein stated, all oxygen insufflation must be extremely gentle. Not only must rupture of the parenchymal walls be avoided but the pressure must not be sufficient to cause ischemia of the parenchymal capillaries. There must be no pressure. There is no need for any. Ample ventilation of the lungs is assured by introduction of the oxygen-carbon dioxide gas into the trachea near the bifurcation, whether there are thoracic respiratory movements or not. With the ordinary oxygen tank, gentleness is secured by maintenance of an ample return flow and by the admission of only one or two bubbles of gas through the water of the wash bottle per second. After aspiration, the infant-size Drinker mechanical respirator may be used, if available, to carry on respiration until the infant can do his own breathing, but if there is obstruction in the airway, removal of obstruction is the first step without which nothing can be helpful.

Another form of neonatal asphyxia is that due to *congenital bilateral laryngeal paralysis*. The cause of the paralysis in the cases we have seen was probably pressure on the central or peripheral laryngeal abductor neurons during accouchement. All were cases of prolonged difficult primiparal labors necessitating instrumental delivery. All the infants made thoracic movements but could get almost no air. The skin was bluish. The laryngoscope revealed the vocal cords remaining in the midline, causing

inspiratory stridor. The bronchoscope was inserted, the cyanosis disappeared, and the breathing through the bronchoscope became quiet and regular. The insufflation tube was substituted for the bronchoscope. The paralysis disappeared spontaneously the following day. It is not necessary to use the bronchoscope in these cases, the insufflation tube is sufficient. The bronchoscope was used for diagnosis in the cases mentioned because thymic compression was suspected. There was no bulbar paralysis in these cases. If there had been, the use of the Drinker infant-respirator would have been rendered possible by the insufflation tube in place admitting air through the otherwise tightly closed glottis. It is characteristic of mid-line laryngeal paralysis that inspiration, normal or artificial, sucks the cords tightly together for anatomic reasons elsewhere herein explained (Fig. 357).

It is important to distinguish between *bulbar respiratory paralysis* and *abductor laryngeal paralysis*, as elsewhere mentioned. The bulbar form, with unimpaired laryngeal movement, is likewise amenable to treatment by intratracheal insufflation as a temporary expedient, but, of course, for long duration nothing is equal to the Drinker apparatus.

For impending *neonatal asphyxia due to drugs* given the mother, intratracheal insufflation as above described will prevent asphyxia until the drug is eliminated, but after endoscopic clearing of any obstruction in the airway in such cases the Drinker infant apparatus should be used if available.

In concluding the subject of asphyxia neonatorum the writer wishes to repeat for emphasis that the utmost gentleness is required in the use of direct methods of clearing the airway and getting oxygen into the tissues. These methods will go a long way in reducing the high mortality in these cases, but the tender infant, just out of his comfortable life afloat, requires gentle handling.

Prognosis in Impending Asphyxia.—When impending asphyxia is due to curable conditions the prognosis depends on the availability of equipment and trained personnel. The "Society for the Prevention of Asphyxial Death" has been organized and founded by Pafuel J. Flagg. It should go a long way in improving the prognosis of impending asphyxia.

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Tracheotomy is the operation of making an airway through the neck into the trachea. It is usually done to prevent asphyxia. Former distinctions between high and low tracheotomy, based on the location relative to the thyroid isthmus, are now obsolete.¹ There are two types of procedure, orderly tracheotomy and emergency tracheotomy.

Orderly Tracheotomy.—*Indications.*—Tracheotomy is indicated when the cardinal signs of obstructive laryngeal dyspnea (*qv*) are definitely present. Deferment is justified only when the patient is under constant observation and a competent tracheotomist is promptly available. The many morbid conditions requiring the operation are considered in connection with the respective diseases.

Contraindications.—There are no contraindications. With an expert intubator on twenty-four-hour duty intubation may be preferable in diphtheria, but not otherwise. In all other acute conditions tracheotomy is preferred by most authorities. As mentioned in the preceding paragraph tracheotomy may be postponed, but usually the postponement is regretted.

Instruments.—The modern cannula is made without the projections that formerly increased its bulk, entangled gauze in the wiping away of secretions, and prevented the proper occlusion by the patient's finger after the wound had healed (Fig. 359). It is made in eight standard sizes and in many special forms for special purposes. A proper cannula seems too long when first inserted but extra length is taken up by swelling of the soft tissues which would lift a shorter cannula out of the tracheal slit and

thus cause dyspnea or even risk asphyxia. The extra length allows for ample dressings to absorb secretions in the postoperative period, it can be shortened for subsequent wear if desired.²

The following is a list of instruments for tracheotomy.

- 1 Tracheal cannulae (Fig. 359). Size no. 5 is suitable for adults, 4 for adolescents, 2 and 3 for children, 1 for infants. Duplicate (exact) will be needed in the after care.
- 2 Scalpels (Fig. 360).
- 3 Curved, probe pointed bistoury.
- 4 Tenaculum special Chevalier Jackson model.
- 5 Two small retractors.
- 6 Trousseau dilator.
- 7 Six hemostats (special model preferable).
- 8 Scissors (dissecting).
- 9 Full curved needles very small for suture ligatures.
- 10 Full curved needles (or clamps) for the skin.
- 11 Needle holder.
- 12 Suture material (or clamps).
- 13 Hypodermic syringe for local anesthesia with bent reinforced needle (special model preferable).
- 14 No. 1 plain catgut ligatures.
- 15 Linen tape.
- 16 Gauze sponges.
- 17 Soft rubber catheter (10 F) for aspiration of the trachea through the cannula.
- 18 Aspirator preferably electrically operated.
- 19 Tank containing oxygen with 7 per cent admixture of carbon dioxide.
- 20 Direct laryngoscopic and bronchoscopic outfit should be sterile and promptly available in case of need.

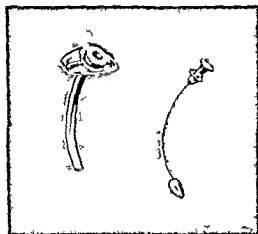


Fig. 359—Modern tracheotomy cannula (C. L. J.). The shield is free from all entangling projections. This cannula is made in eight standard sizes.

Anesthesia—General anesthesia would introduce the risk of precipitating a necessity for emergency tracheotomy because any patient with enough obstruction in his airway to require

tracheotomy is certain to do badly under general anesthesia. Local anesthesia by the technic given as "Step 2" renders the procedure painless. To get the quiet cooperation of the patient he should be assured that the operation is not a serious one, that he will feel no pain and little

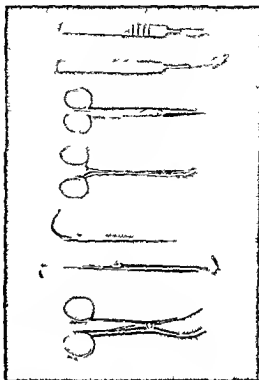


Fig. 360—Instruments for orderly tracheotomy. It is true that in an emergency a tracheotomy can be done and a life can be saved with a knife and a pair of hands, but every hospital and every physician should have an emergency equipment such as this, sterile and packed in a wrapped copper box. Instruments from above downward: scalpel, fistula bistoury, dissecting scissors, hemostat, fixation tenaculum, retractor, Trousseau dilator, copper box.

discomfort, and especially he should be told that the operation will not take long, usually only a few minutes. It is well also to mention the fact that he may at times have his breath shut off, but that you know when this happens and you will not keep it shut off too long.

Technic—The position of the patient on the operating table should be that shown in Figure 361. The sand pillow is sketched in black to emphasize the importance of its placement under the shoulders, not under the neck. The position shown gives greatest prominence to the trachea and maximum access to the front of the neck. If the patient is extremely dyspneic he is not put in this position until the knife is about to make the incision.

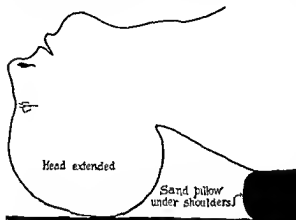


Fig 361—Position of patient for tracheotomy

For clearness the procedure may be described in twelve steps, as follows

Step 1 Local Preparation The previously shaven skin is prepared in the usual way with tincture of iodine followed with alcohol.

Step 2 Local Anesthesia Novocaine solution of standard strength is withdrawn from ampules directly into the hypodermic syringe. First the midline is injected from the hyoid bone to the suprasternal notch, then the deeper tissues of the midline. There is no need of going far laterally because flaps are not to be dissected up. Usually there is no need of any injection after starting. Instilling cocaine into the trachea is inadvisable, it seems better to allow the cough reflex to clear the trachea and bronchi as soon as the trachea is opened.

Step 3 The initial incision in the midline from Adam's apple to the suprasternal notch passes through the skin and superficial fascia. Care is taken in the upper part of the incision to avoid cutting the external perichondrium of the laryngeal cartilages which are usually quite close to the skin. Integumentary vessels are clamped if necessary.

Step 4 Dry Dissection No flaps are dissected up. The incision is deepened in the midline down to the thyroid cricoid, and tracheal cartilages, care being taken not to injure the external perichondrium, and not to lay it bare except in the midline. The larynx and trachea should not be skeletonized. As in any orderly operation a dry wound should be maintained. There are no large arteries in the midline of the neck, but rather large veins may be encountered. They should be caught and cut off squarely between two hemostats. Nicks, slanted cuts, or torn walls are likely to spring a leak later during cough.

Step 5 Disposal of the Thyroid Isthmus The isthmus or even the thyroid gland must not be allowed to interfere with a properly placed incision of the trachea (see Step 8). Sometimes the most that will be necessary is to drag the isthmus upward or downward, but if the isthmus is not atrophic it should be divided by a vertical median cut between two hemostats slipped on from above downward. Each stump may then be ligated external to its hemostatic clamp, or, if large, each end may be quickly closed by a continuous overhead suture. If the isthmus is drawn upward or downward the direction of displacement must be remembered for the finding of the tracheal slit at the first few dressings.

Step 6 Hemostasis Preparatory to the tracheal incision all hemostats should be taken off and a dry wound assured by ligature or torsion. Even slight oozing will result in a spray of bloody droplets at each cough after the trachea is incised.

Step 7 Tracheal Fixation For a precise incision the trachea is first fixed with the tenaculum. As a rule the hook is passed through the interannular membrane at the lower border of the second ring, in the midline. With the hook under, not sticking in, the second ring, the trachea is lifted and thus fixed, its identity is also thus verified.

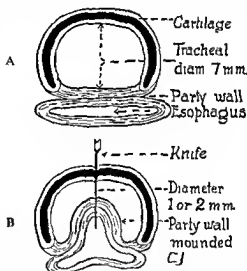


Fig 362—Sketches showing one of the pitfalls in tracheotomy especially in children. In A is shown how the posterior wall of the trachea forms an integral party wall. In B is shown how the cough reflex causes the party wall to mound forward against the knife and thus to perforate into the esophagus. This usually fatal disaster is best avoided by having a bronchoscope in the trachea. Lacking this the middle finger on the knife blade should be used as a depth gauge.

Step 8 Tracheal Incision The trachea is incised in the midline caudad from the hook of the tenaculum through the third, fourth and fifth rings. Caution is necessary to avoid incising the tracheo-esophageal party wall. The tracheal rings are really horseshoe shaped because they are incomplete posteriorly. The membranous posterior wall is especially likely to be cut in children because of the small diameter of the trachea and especially because this small diameter is still further reduced by the mounding forward of the posterior wall during cough (Fig

362) In adults the posterior wall does not mound forward much because of greater rigidity of the tracheal cartilages, but the accidental puncture into the esophagus is especially likely to happen from the plunging of the knife that has been pressed upon in order to cut through the partially ossified rings. Both these accidents are avoided by guarding the knife with the finger. If preferred, only one ring may be cut with the scalpel and the others then cut with the probe-pointed, curved bistoury.

Step 9 Spreading the Tracheal Incision. Though there may be a hiss of air as the trachea is incised, the edges of the incision usually approximate so tightly that little air passes. The Trousseau dilator is inserted and the tracheal incision spread. Almost always there is a clot of mucus expelled. If the dyspnea has been considerable the patient takes a deep breath that ends in a sigh of relief. A child worn out by a prolonged fight for air may fall asleep on the operating table.

Step 10 Insertion of the Cannula. To make sure the cannula enters the trachea the piloted distal end must be seen to pass in through the tracheal incision. The pilot is usually blown out by the tussive blast, if not, it should be quickly removed. The shield of the cannula, if the tube is of proper length, should stand out a little from the level of the skin.

Step 11 Closure of the Wound. This may be done either with interrupted sutures or with clamps, but the most important thing is that the wound must not be closed completely, that is, not close up to the cannula. It is better to close only a short part of the upper and lower ends of the incision. (For reasons see under Difficulties, Pitfalls, and Complications of Tracheotomy.)

Step 12 Dressing and Drainage. With an open wound no drainage is necessary. A pad made of six or more layers of folded gauze is split halfway so as to go down astride the cannula back of the shield. Strips or small pieces of gauze are to be avoided because of the danger of an end getting into the trachea or cannula.

After Care.—At the top of the order sheet is written "Opiates or atropine should not be given this patient."^{3, 4} The patient is put to bed with the usual postoperative nursing attention. If the nurse has not had good tracheotomic training she must be instructed in the signs of dyspnea and in the quick but gentle wiping away of the coughed out secretions before they are inspired into the cannula. She must be warned that the patient is voiceless, consequently cannot call for help, or, in case of a baby, cry audibly. Dehydration must be avoided. As no nauseating anesthetic or sedative has been given the patient may begin swallowing liquid food at any time. It is exceedingly rare that a feeding tube is required, but there is no contraindication to using it. The room should be well ventilated. In summer this will insure sufficient humidity in most climates. In winter, in the United States the air will require humidification. The outdoor air at, say

zero, contains almost no water, when this air is heated to 70° F it becomes almost caustic to the tender tracheal mucosa of a child. Moistening of the air should be done without raising the temperature over 70° F because this defeats the object, by simultaneously raising the water-absorbing power of the air. Mechanical humidifiers are best for this reason. A dusty atmosphere is to be avoided.

In the local care the keynote is "good plumbing." That is to say, every moment throughout each twenty four hours there must be present someone who understands the necessity for, and the means of maintaining clear "pipes," natural and artificial. And this person must understand that tracheotomy is not an ultimate object. The objective is to pipe air down to the lungs, and tracheotomy is only a means to that end. Secretions must not be allowed to accumulate and coagulate in the trachea, if they are not coughed out they must be aspirated. If crusts form they must be removed by bronchoscopy, sometimes coagula are so firm as to require forceps removal.

A bedside tray should contain the following: (1) sterile gloves, (2) sterile gauze, (3) scissors, (4) probe, (5) two hemostats, (6) Trousseau dilator, (7) duplicate (exact) tracheotomic cannula, (8) infant sized catheter for aspiration, (9) a doubled 2-foot length of soft copper wire for pulling gauze through cannulae for cleansing before boiling (wire gauge no. 24 is heavy enough, tonsil snare wire should not be used because it will damage the thin edge of the inner end of the cannula).

A mechanical aspirator and a tank containing oxygen (plus carbon dioxide, 7 per cent) should be in the room, ready for immediate use. A bronchoscope, bronchoscopic forceps, cords, battery, and aspirating tube should be quickly available. Dressings are to be changed as frequently as soiled (usually every few hours). This has the effect of keeping the wound washed with sterile exuded serum, which is removed at each change of dressings. The inner cannula is removed, cleansed in lukewarm sterile water, and promptly replaced. The outer cannula should be changed within six hours after operation to get rid of clots and inspissated secretions, afterward at least once daily. There should be duplicate cannulae and they should be exact duplicates, so the inner cannula of either may be used in the changing of inner cannulae, and so the duplicate outer cannula can

be ready at all times for immediate substitution. Not only is delay avoided, but also the hasty cleaning that is so likely to be imperfectly done, with resultant boiled secretions in the lumen and damage to the inner end of the cannula. A damaged inner end causes endotracheal mucosal trauma and crusting, and it may lead to perichondritis of the tracheal rings. The patient, if over two years of age, may be alarmed at inability to speak. He should be assured that this is only because the air leaks out

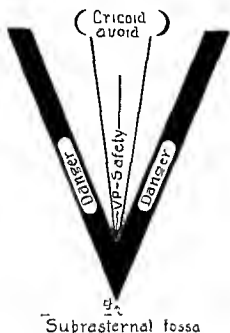


Fig. 363—Schema of practical gross anatomy that should be memorized by every medical student. The midline of the neck is the safety line; the higher the wider. Below, the safety line narrows to the vanishing point VP. The upper limit of the safety line is the thyroid notch until the trachea is bared when the lumen falls below the first tracheal ring. In practice the trunks are pushed back under danger lines with left thumb and middle finger (Fig. 364) thus throwing the safety line into prominence. This is generally known as Chevalier Jackson's tracheotomic triangle.¹

through the cannula, and that in about ten days he can talk as well as ever by putting his finger on the cannula. This may be demonstrated to him by the surgeon occluding the cannula momentarily, at a dressing. The patient, if there are no complications or general contraindications, may sit up on the third or fourth day and go about at the end of a week. The wound may not be quite healed by that time, but there is then no danger of serious pyogenic infection.

Emergency Tracheotomy—The best procedure, in dealing with an asphyxiating patient, is

to insert a bronchoscope, establish regular respiration through it, and then perform a low tracheotomy in an orderly manner. In addition to elimination of hasty tracheotomy, this technic has the advantage of direct laryngoscopic and bronchoscopic inspection of the living pathologic tissue changes present in the particular case. Additionally, the bronchoscope in situ in the trachea makes the operation quite simple, by affording a staff that may be cut down upon. The conversion of an emergency into an orderly procedure can also be accomplished by use of Mosher's "life savers." These are curved tubes with a long straight shank for insertion through the mouth into the larynx by the same motions used in O'Dwyer's blind method of intubation for laryngeal diphtheria, though the instrument itself is quite different (Fig. 352). If, however, neither the bronchoscope nor the life saver is available an emergency tracheotomy is done by the emergency technic. The quickest method that is certain in its results is the Chevalier Jackson two-incision operation. It is done as follows:

Anesthesia—Any patient needing an emergency tracheotomy does not need local anesthesia, and a general anesthetic would be promptly fatal.

Instruments—A knife and a pair of trained hands are enough, but a full surgical equipment as listed on page 479 is better.

Position of the Patient—The neck should be thrown into prominence by pushing a roll of fabric of any kind under the shoulders, not under the neck. If the patient is on the floor or pavement, or in a vehicle, the head may be lifted and the neck brought above the left knee of the kneeling or, preferably, sitting operator.

Technic—For rapid performance it is well for the operator to ignore the details of anatomy and of orderly procedure previously described and to obtain instead a mental conception of the tracheotomic triangle (Fig. 363). He should get the conception of a windpipe in the midline covered by soft tissues through which he must cut, and can cut safely, so long as (1) the patient's head is not rotated, (2) the cutting is in the midline, (3) the operator's left index finger is educated to recognize the feel of the trachea, (4) the trachea is thrown into prominence between the operator's left thumb and middle finger. The throwing of the trachea into prominence should be rehearsed at every opportunity, such as on a member of the surgeon's

family, a student, orderly, or any patient whose neck is being examined. It is the most important feature of the procedure.

For clearness of description and fixation in the memory the procedure may be divided into seven steps, but in practice the steps merge into each other so rapidly and smoothly that it seems but a single procedure, and it requires less than a minute.

Step 1 Throwing the trachea into prominence and fixing it in the midline are accomplished by pushing back the great vessels well under the sternomastoid muscles. This is done in a second or two with the thumb and index finger of the left hand (Fig. 364 A).

Step 2 Initial Incision. With one sweep of the scalpel an incision is made from Adam's apple almost to the suprasternal notch. This incision must go clear through

scalpel in the right hand can be safely guided down along the palmar surface of the index finger onto the tracheal wall. Two or three rings are incised. In making the incision the middle finger of the right hand makes a guard against deep entrance of the knife that might otherwise go through the posterior tracheal wall into the esophagus (Fig. 363).

Step 3 Holding Apart the Lips of the Tracheal Incision. There is usually a hiss if the patient is breathing, but the lips of the tracheal incision in most cases lie so closely in contact that insufficient air is permitted to pass. The lips should be spread apart with a Trousseau dilator, if none is available a hemostat will do. Lacking these, the handle of the scalpel may be inserted in the slit and rotated slightly to cause gaping. Of course some blood will trickle into the trachea but cough will quickly expel it and spray it about. The cannula is inserted with its pilot; the pilot is removed if not blown out by the tracheal blast. If by reason of deplorable lack of equipment no cannula is available someone must hold the lip of the

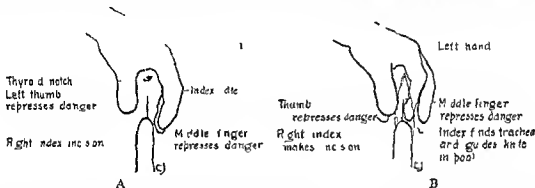


Fig. 364—A, Sketch showing emergency tracheotomy by the two-incision finger dissection technique. The large vessels are pressed back under the respective sternomastoid muscles with the thumb on one side and the middle finger on the other. This repression of the danger lines at the same time throws the trachea into prominence. With the notch of the obvious thyroid cartilage as a landmark the midline of the neck is slashed through skin and subcutaneous tissues at the first incision.

B. The danger lines being still repressed the previously idle left index finger burrows by sense of touch down along Adam's apple over the cricoid membrane and two rings of the trachea, easily felt as corrugations. The knife slides in along the palmar surface of the index finger to make the tracheal incision. This completes the emergency tracheotomy in two incisions.

the skin to facilitate free finger dissection. There may be considerable flow of blood but nothing immediately dangerous.

Step 3 Finger Dissection to Find the Trachea (Fig. 364 B). While the left thumb and middle finger are still holding back the great vessels under the sternomastoids the previously idle left index finger quickly locates the trachea by following downward from Adam's apple. The trachea is infallibly identified by the ridgy character of its walls; there is nothing like it in that region. Every medical student has learned or should have learned the feel of it in the dissecting room. In identifying the trachea the finger at the same time bares its midline of tissue below the cricoid by pushing the finger down under any overlying tissue; if the isthmus of the thyroid gland intervenes it is pushed downward and elevated. If it is torn or cut it will not matter. If an anomalous artery is felt crossing the trachea at this point it is pushed downward.

Step 4 Tracheal Incision. The trachea having been identified and bared of tissue the tip of the index finger is slid slightly to the operator's left side so that the

tracheal incision apart until a cannula can be obtained or a makeshift devised for temporary use.

Step 6 Artificial respiration must be done if there has been respiratory arrest. Oxygen mixed with 7 per cent carbon dioxide should be liberated at the tracheal opening or gently insufflated through a catheter. The best cardiac stimulant is to get oxygen into the heart muscle by way of the circulating blood. No drug is a substitute for this.

Step 7 Hemostasis. As soon as respiration is established hemostasis may be done in the usual way. If artificial respiration is necessary temporary hemostasis may be afforded by packing gauze firmly into the wound and around the cannula.

The dressings and after care are the same as given for orderly tracheotomy, except that, when emergency has necessitated disregard of aseptic technique, it is advisable to dust the wound lightly with any available sulfonamide at the first few dressings. A total of 0.1 gm., locally at a dressing is safe even in children. The cannula can be momentarily occluded to prevent inspiration of uncontrolled amounts of the powder.

be ready at all times for immediate substitution. Not only is delay avoided, but also the hasty cleaning that is so likely to be imperfectly done, with resultant boiled secretions in the lumen and damage to the inner end of the cannula. A damaged inner end causes endotracheal mucosal trauma and crusting, and it may lead to perichondritis of the tracheal rings. The patient, if over two years of age, may be alarmed at inability to speak. He should be assured that this is only because the air leaks out

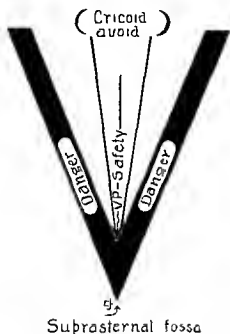


Fig. 363—Schema of practical gross anatomy that should be memorized by every medical student. The midline of the neck is the safety line; the higher the wider. Below, the safety line narrows to the vanishing point VP. The upper limit of the safety line is the thyroid notch until the trachea is bared, when the limit falls below the first tracheal ring. In practice the trunks are pushed back under danger lines with left thumb and middle finger (Fig. 364) thus throwing the safety line into prominence. This is generally known as Chevalier Jackson's tracheotomic triangle.¹

through the cannula, and that in about ten days he can talk as well as ever by putting his finger on the cannula. This may be demonstrated to him by the surgeon occluding the cannula, momentarily, at a dressing. The patient, if there are no complications or general contraindications, may sit up on the third or fourth day and go about at the end of a week. The wound may not be quite healed by that time, but there is then no danger of serious pyogenic infection.

Emergency Tracheotomy—The best procedure, in dealing with an asphyxiating patient, is

to insert a bronchoscope, establish regular respiration through it, and then perform a low tracheotomy in an orderly manner. In addition to elimination of hasty tracheotomy, this technic has the advantage of direct laryngoscopic and bronchoscopic inspection of the living pathologic tissue changes present in the particular case. Additionally, the bronchoscope in situ in the trachea makes the operation quite simple, by affording a staff that may be cut down upon. The conversion of an emergency into an orderly procedure can also be accomplished by use of Mosher's 'life savers'. These are curved tubes with a long straight shank for insertion through the mouth into the larynx by the same motions used in O'Dwyer's blind method of intubation for laryngeal diphtheria, though the instrument itself is quite different (Fig. 352). If, however, neither the bronchoscope nor the life saver is available, an emergency tracheotomy is done by the emergency technic. The quickest method that is certain in its results is the Chevalier Jackson two incision operation. It is done as follows:

Anesthesia—Any patient needing an emergency tracheotomy does not need local anesthesia, and a general anesthetic would be promptly fatal.

Instruments—A knife and a pair of trained hands are enough, but a full surgical equipment as listed on page 479 is better.

Position of the Patient—The neck should be thrown into prominence by pushing a roll of fabric of any kind under the shoulders, not under the neck. If the patient is on the floor or pavement, or in a vehicle, the head may be lifted and the neck brought above the left knee of the kneeling or, preferably, sitting operator.

Technic—For rapid performance it is well for the operator to ignore the details of anatomy and of orderly procedure previously described and to obtain instead a mental conception of the tracheotomic triangle (Fig. 363). He should get the conception of a windpipe in the midline covered by soft tissues through which he must cut, and can cut safely, so long as (1) the patient's head is not rotated, (2) the cutting is in the midline, (3) the operator's left index finger is educated to recognize the feel of the trachea, (4) the trachea is thrown into prominence between the operator's left thumb and middle finger. The throwing of the trachea into prominence should be rehearsed at every opportunity, such as on a member of the surgeon's

family, a student, orderly, or any patient whose neck is being examined. It is the most important feature of the procedure.

For clearness of description and fixation in the memory the procedure may be divided into seven steps, but in practice the steps merge into each other so rapidly and smoothly that it seems but a single procedure, and it requires less than a minute.

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scalpel in the right hand can be safely guided down along the palmar surface of the index finger onto the tracheal wall. Two or three rings are incised. In making the incision the middle finger of the right hand makes a guard against deep entrance of the knife that might otherwise go through the posterior tracheal wall into the esophagus (Fig. 363).

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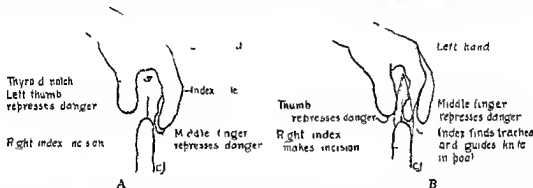


Fig. 364—A, Sketch showing emergency tracheotomy by the two incision, finger dissection technique. The large vessels are pressed back under the respective sternomastoid muscles with the thumb on one side and the middle finger on the other. This repression of the danger lines at the same time throws the trachea into prominence. With the notch of the obvious thyroid cartilage as a landmark the midline of the neck is slashed through skin and subcutaneous tissues at the first incision.

B, The danger lines being still repressed the previously idle left index finger burrows by sense of touch down along Adam's apple over the encothyroid membrane and two rings of the trachea, easily felt as corrugations. The knife slides in along the palmar surface of the index finger to make the tracheal incision. This completes the emergency tracheotomy in two incisions.

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Step 3 Finger Dissection to Find the Trachea (Fig. 364 B). While the left thumb and middle finger are still holding back the great vessels under the sternomastoids the previously idle left index finger quickly locates the trachea by following downward from Adam's apple. The trachea is infallibly identified by the ridgy character of its walls, there is nothing like it in that region. Every medical student has learned or should have learned, the feel of it in the dissecting room. In identifying the trachea the finger at the same time bares its midline of tissue below the cricoid by pushing the finger down under any overlying tissue. If the isthmus of the thyroid gland intervenes it is pushed downward and elevated. If it is torn or cut it will not matter. If an anomalous artery is felt crossing the trachea at this point it is pushed downward.

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tracheal incision apart until a cannula can be obtained or a makeshift devised for temporary use.

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The dressings and after care are the same as given for orderly tracheotomy, except that, when emergency has necessitated disregard of aseptic technique, it is advisable to dust the wound lightly with any available sulfonamide at the first few dressings. A total of 0.1 gm. locally, at a dressing is safe even in children. The cannula can be momentarily occluded to prevent inspiration of uncontrolled amounts of the powder.

Difficulties, Pitfalls, and Complications of Tracheotomy.—If the technic has been closely followed there will be no serious difficulty. Departure from the technic involves risk of pitfall. A common one is the attempt to give a general anesthetic. Dyspneic patients are dependent largely on the aid of the accessory muscles of respiration under voluntary stimulus. This is abolished as soon as loss of consciousness begins, respiration ceases and unless bronchoscopy or tracheotomy is done within two or three minutes the respiration cannot be started and the patient asphyxiates. In the aggregate the mortality from this pitfall has been enormous. General anesthetic should never be used for tracheotomy unless a bronchoscope is in situ, and there is no need for it even then. It may be added that opiates for the restlessness of air hunger in children needing tracheotomy have often precipitated an emergency tracheotomy, and in many cases have resulted in death by asphyxia.^{3, 4} A short incision is a common pitfall. Complete closure of the wound close up to the cannula, is a pitfall of not infrequent occurrence. A false passage by the cannula going astray may occur at any of the early dressings, and lead to a slow asphyxia. With an open wound the tracheal incision can be found with the Trousseau dilator and the cannula can be seen to enter the trachea. Mediastinal emphysema is usually due to stitching the wound close up around the cannula. The air in the cervical tissues works downward between the layers into the mediastinum. This pitfall can be avoided by packing the wound open as previously described. Rarely the air may leak into the pleural cavity causing a pneumothorax. Spontaneous pneumothorax has occurred from rupture of the visceral pleura in violent respiratory efforts in impending asphyxia.⁴ This may be favored by old adhesions. Unilateral pneumothorax in such cases is rarely followed by empyema and the lung re expands normally. Bilateral pneumothorax is quickly fatal unless bronchoscopic oxygen insufflation is promptly instituted and constantly maintained until at least one lung has expanded. Pneumothorax may occur from direct trauma to the pleura in an excessively low tracheotomy. Pneumonia after tracheotomy is one of the curiosities of medicine. The frequent mention of this complication in the literature is due to mistakes of diagnosis. The often fatal pulmonary condition is really an obstructive atelectasis completely re-

lievable by bronchoscopic methods. Hundreds of times we have had it reported to us that our tracheotomic patient was "dying of pneumonia." Prompt bronchoscopy almost invariably revealed plugs of thick secretion, or crusts, or both, obstructing bronchial orifices. Bronchoscopic removal caused disappearance of the fever, high respiratory rate, and impaired percussion note on which the erroneous diagnosis of pneumonia had been based. Auscultation is not so misleading as percussion in these cases. Erysipelas and serious pyogenic infections are rare complications. If they should occur the sulfonamides or penicillin should be used (see under Chemotherapy in Otolaryngology).

Laryngeal and tracheal stenoses are usually due either to a high tracheotomy or a misfit cannula, two pitfalls that are easily avoided. Tracheotomy should be performed below the second ring of the trachea. If this is done there will be no stenosis caused by the tracheotomy.¹ All patients requiring prolonged wearing of a tracheotomic cannula should have the cannula checked for perfect fit. A misfit cannula will cause erosion of the tracheal mucosa, perichondritis, and chondral necrosis. If roentgen ray examination shows the cannula has too short or too long a radius of curvature a perfect fitting cannula must be made from measurements best obtained with the measuring apparatus of Chevalier L. Jackson.² Stenosis also occurs from destructive processes of the primary disease, for example diphtheria, syphilis, tuberculosis, typhoid fever. When the cartilaginous framework is destroyed even partially, the unresisted cicatricial contraction will obliterate the laryngotracheal lumen. This must be frustrated and the lumen restored by the early use of core molds.

Decannulation—The importance of proper procedure in decannulation is not always realized and this sometimes leads to disastrous consequences. In case of a child, simply taking out the cannula often creates panic because breathing through a cannula in the neck is unobstructed compared to the circuitous route past the natural 'baffle plates' in the nose, nasopharynx, and pharynx. Moreover, there is no certainty that an abundant airway has been obtained. A test must be made. The first step is to make certain the cannula is as small as it should be. That is small enough in diameter to allow for by passage of air in the trachea, when the cannula is corked. If necessary a

smaller size may be substituted. The next step is partial occlusion of the cannula with a partial cork, usually a half-cork (Fig 367). After the child can sleep soundly and comfortably, for say, three nights with the half-cork it is replaced with a three-quarter cork. When this can be comfortably worn for a like period the seven-eighth size is substituted. The final test is with the whole cork. When this can be worn night and day (with daily toilet, of course), the cannula may be removed and the fistula may be allowed to close. If the fistula is epithelialized and hence leaks secretion it can be closed as described under cicatricial stenosis (Fig 376).

If dyspnea and restlessness develop from an increased size of cork it can be replaced for the night with the previously used size. The daily wearing of the last size will enlarge the lumen so that it soon can be worn at night. If the child cannot be decannulated in a few weeks, treatment for stenosis will be needed. The methods are given on a subsequent page.

CHEVALIER JACKSON

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TUBERCULOSIS OF THE LARYNX

Tuberculosis of the larynx is a specific disease of the larynx due to infection with *Mycobacterium tuberculosis*.

Tuberculosis is one of the commonest chronic specific infective diseases of the larynx. Statistics vary widely as to the relative incidence in cases of pulmonary tuberculosis. Many series of autopsies show that not more than 20 per cent of persons whose death was due to chronic pulmonary tuberculosis show evidence of laryngeal involvement. The clinicians, however, dealing with all stages of the pulmonary disease, do not find more than 10 per cent with laryngeal involvement and some phthisiologists say the incidence of this complication in their clinical experience is not more than 3 per cent. It is certain that the severe cases of laryngeal tuberculosis, that were so numerous in the experience of the older laryngologists, are rare today. The inference that this decreased incidence is due to improved treatment of pulmonary tuberculosis seems justifiable.

There are three distinct clinical types of the disease, the chronic, the acute military, and the lupoid. Of these only the chronic is common.

Etiology.—The primary cause of the disease is the *Mycobacterium tuberculosis*. Secondary invasion by mixed infections causes much of the destructive pathologic tissue changes. Among the secondary invaders may be mentioned streptococci, staphylococci, and spirochaetae. There is another exciting cause of the utmost importance, namely, vocal abuse. Dust, smoke, inhaled tobacco smoke, coughing, and other forms of local trauma contribute to infective processes. The chief predisposing cause may be said to be pulmonary tuberculosis, inasmuch as the laryngeal disease appears practically always in persons affected with the pulmonary disease. Back of that there are all the well-known causes of pulmonary tuberculosis. The systemic effects of deficient sunlight, foul atmosphere, overwork, tobacco, and alcoholic beverages lessen resistance to infection of the larynx, just as they do to pulmonary infection. Laryngeal involvement is recorded as more frequent in the second and third decades, but it may occur at any age.

Pathology.—The histologic tissue changes in tuberculous lesions of the larynx are the same as in other soft tissues with mucosal surfaces and neighboring cartilages. The first stage is

one of anemia. Then follows a pale swelling that resembles edema but is a cellular infiltration, not a serous exudation. Nodules, not always visible on the surface, develop as the result of accumulation of migrating connective-tissue cells and the proliferation of vascular endothelial cells, all of which surround the invading tubercle bacilli in the form of a defensive barrier. In the center of the nodule, cellular delineation slowly disappears, the nuclei lose their staining affinity and scattered giant cells appear as caseation develops. Such nodules may be

one or both of these cartilages may slough out as sequestra, or the joint may become fixed. The lumen of the larynx may be encroached upon by (1) the tuberculous lesion, (2) edema, (3) median fixation of the cords due to tuberculous arthritis of the crico-arytenoid joints, or (4) cicatricial contraction. If ulcers heal before the perichondrium is reached there may be extensive scarring, but it seldom causes the degree of stenosis that results from cicatricial contraction unresisted by an undamaged cartilaginous framework. Motionless glottic



Fig. 365.—Tuberculosis of the larynx

numerous and they have a strong tendency to coalesce into larger lesions. Further progress may be in one of two directions. The defensive process may be effective, the bacilli may become sealed up in a fibrotic capsule, or the caseated nodules may break down and discharge on the surface, secondary pyogenic invaders then swarm in, and a spreading ulcerative process reaches the perichondrium. Perichondritis and necrosis of cartilage are processes of maximum destruction. In this stage there is often a true edema. The perichondritis frequently affects the arytenoid cartilages first,

margins may be due to (1) infiltration of the marginal tissues, (2) total loss of the cordal supportive pyramid, (3) infiltration of the intrinsic muscles, (4) infiltration of the crico-arytenoid joints, or (5) paralysis of the inferior laryngeal nerve by neuritis, or by pressure of a tuberculous node or an infiltrated pleura or a tuberculous pulmonary apex.

Reflected Appearances—The image in the mirror depends upon the stage of the disease. In the earliest stage, tuberculous nodules below the surface may be developing yet the mucosa may be smooth and anemic. In many cases at

this early stage there is a redness of one cord that contrasts with the anemic mucosa. In some cases there is roughening or erosion of one or both cords. As the tuberculous process nears the surface, swelling is noticeable. It may be red but is often normally pink in color, sometimes whitish or bluish pink. The interarytenoid space is the most frequent site of this nodular swelling, but it may appear anywhere. At a later stage, which might be called the second, the aryepiglottic folds look evenly edematous, and the epiglottis may show a similar condition. This is the characteristic picture so often described as pyriform arytenoids and turban-shaped epiglottis. In a third stage there may be ulceration and nodules as well as infiltration and irregularity in any region. The inflammatory reaction to mixed infections may cause redness surrounding the ulcerated areas. The image in the fourth stage is composed of pallor, edema, ulceration, tuberculomas, exuberant granulations often resembling papillomas, obliteration of landmarks, a notched epiglottis, immobility, and a small glottic chink. Any one or more of these features may be present or absent or hidden from view in the mirror. This is the stage of perichondritis and chondral necrosis.

Appearances on Direct Laryngoscopy.—All of the appearances described in the preceding paragraph, and more or less visible in the mirror, come strongly and clearly into view in the laryngoscope. The regions hidden under the overhang of the epiglottis and of the ventricular bands, and any masses of diseased tissue, are all brought into line of direct vision. The presence or absence of cords can be determined and differential diagnostic points can be definitely decided. When palpated with the closed laryngeal forceps used through the direct laryngoscope the enlarged aryepiglottic folds are found to be firm tissue infiltrations, not truly edematous, as they seemed to be in the mirror. The passive mobility test will promptly determine presence or absence of any involvement of the crico-arytenoid joints or of their attachments.

Symptoms.—The first laryngeal symptom, often present long before there is any suspicion of even a pulmonary tuberculous lesion, is weakness of the voice. Next comes slight hoarseness usually in intermittent attacks often attributed by the patient to "catching cold." A slight cough may be noted. In the next stage

the hoarseness is intermittent in degree but does not disappear altogether in the remissions. Cough is more annoying and is slightly productive. The next stage is accompanied by a wheezy cough, blood streaked sputum, dyspnea, dysphagia, odynophagia, odynophonia, and aphonia. Pain in the ear is a frequent complaint and neuralgia of the superior laryngeal nerve occurs in a few cases. The foregoing refers only to laryngeal symptoms. They may be masked by the pulmonary symptoms especially when laryngeal involvement appears in an advanced stage of the pulmonary disease.

Diagnosis.—Though it is true that laryngologists of long experience may often make the diagnosis of laryngeal tuberculosis on first inspection, it is also true that they may occasionally make serious errors, in both negative and positive opinions. It is best for the patient's welfare to take the appearances in the mirror simply as a working diagnosis while studying the case. There are two classes of cases, according to whether a diagnosis of pulmonary tuberculosis is beyond question or is entirely unsupported. In the case of a patient having no evidence of pulmonary disease, the blood Wassermann reaction being negative, an ulcerative or nodular lesion in the larynx would call for immediate taking of a specimen for biopsy, but if the Wassermann reaction is positive the larynx should be kept under observation until a therapeutic test for syphilis is made (see "Syphilis of the Larynx"). If the antisyphilitic treatment is without decisive effect on the laryngeal lesion, the specimen should then be taken without delay. If the patient has developed a weak voice with no apparent lesion in the larynx he should be put on a regimen of silence (*q v*) while being kept under observation, and if in addition to the weak voice he has a dry hacking cough he should be put, additionally, on an antituberculous regimen even if the pulmonary examination has revealed no evidence of tuberculosis. If the weak voice should develop in a patient with definitely tuberculous lesions in the lung, the patient should be treated as one who has incipient laryngeal tuberculosis. In any patient, one persistently red cord should be suspected of tuberculosis, and the anterior commissure laryngoscope should be used to search not only below the cord but in the ventricle, beneath the posterior commissure, and everywhere else within the larynx as well as in the hypopharynx. Often in such cases a

lesion justifying the taking of a specimen, hut invisible in the mirror, will be found. The cord itself may be so obviously diseased that a specimen should be taken by scalping off diseased tissue, even though the cord looked thin and smooth in the mirror. It must always be borne in mind that in adults almost any laryngeal lesion may be tuberculous, syphilitic, or cancerous, and it is always best to suspect cancer until the lesion is proven not to be malignant. Two or even all three of these diseases may coexist (See under "Cancer of the Larynx"). Diagnostic delay is dangerous. It may be added that neither the Von Pirquet nor the old-tuberculin test is of any practical value in the clinical diagnosis of laryngeal disease.

Prophylaxis.—Silence is the best means of prevention of laryngeal tuberculosis. Even though the infection may be conveyed from the lungs or the blood stream, one has only to witness the beneficial effect of a regimen of silence on a well developed tuberculous lesion of the larynx to realize that this regimen of rest is of prophylactic value. Every patient with pulmonary tuberculosis should be treated as one of incipient laryngeal tuberculosis, even though there is no evidence whatever of laryngeal involvement. The general measures for the prevention of pulmonary tuberculosis apply to the larynx but are so well known that they need not be repeated here.

Treatment.—The most powerful factor in the treatment of laryngeal tuberculosis is rest of the larynx, and the best way to obtain this rest is by a regimen of silence (see under "Myasthenia Laryngis")—it is worse than useless to tell a talkative patient not to talk so much. Next in importance comes the general antituberculous regimen. This is well known to every medical graduate. From the viewpoint of laryngeal care a dust-free, not too dry climate where it is pleasant to be outdoors in the maximum of sunshine and skyshine, would be ideal, but the selection of climatic and every other feature of treatment should be determined on the basis of what is best for the pulmonary condition.

Local treatment other than the regimen of silence is not required in every case, though there are no objections to the use of any non irritating spray or inhalation. Galvanopuncture is useful in reducing the pale cellular infiltrations that look so much like edema. The utmost precision and conservatism are necessary to avoid ruin of the laryngeal motility. The lar-

yngeal mucosa is locally anesthetized as described for direct laryngoscopy. An electrode with fine point is accurately placed to avoid the cords, the motor areas (joints and muscles), and the superficial cartilaginous areas. The current previously adjusted for proper heat is turned on by a momentary touch of the switch knob in the cautery handle, and at the same moment the electrode is pushed in gently and with utmost care not to go deeply enough to reach the perichondrium of any cartilage.⁵

For ulcerative tuberculosis of the larynx Lukens' method of treatment of laryngeal tuberculosis has stood the test of time. With the Lukens' syringe (Fig. 341) a few drops of a dilution of Burmese chaulmoogra oil (15 per cent) in liquid petrolatum, are instilled into the larynx under guidance of the image reflected in the mirror. It is necessary to see the drops enter the larynx because, though not irritating in the larynx, chaulmoogra oil is intensely so to the gastric mucosa. If the lesions extend to the extrinsic area, accuracy and minimal quantity are essential to prevent any of the oil reaching the stomach. Odynophagia is an important symptom calling for treatment not only because distress interferes with the general rest essential for treatment of pulmonary tuberculosis, but even more because painful swallowing interferes seriously with the intake of the food that is fundamental to the treatment of any tuberculous condition. The Lukens' treatment with chaulmoogra oil, in the course of a few weeks of biweekly applications, is efficient in relieving this odynophagia as well as in promoting healing of the lesion.

Larocaine in 10 per cent solution, applied in the same way as in the preparation for direct laryngoscopy, will give relief but only for the duration of the meal. This is not practicable except in a sanatorium. As it is not toxic and not irritating to the stomach an intelligent patient or a dependable member of his family might be trusted to drop a definite quantity, say, 0.5 cc (8 minims) down back of the tongue ten minutes before meal time. Larocaine can also be used in the form of a troche (10 per cent in any neutral gum). Cocaine and morphine are the most efficient analgesics, but their habit-forming consequences are so deplorable that their use should be limited to utterly hopeless cases. Moreover, they destroy appetite, an effect particularly deplorable in tuberculosis. On theoretic grounds, it seems possible that

chemotherapy (*q. v.*), might be useful in the control of the mixed pyogenic infections that cause so much destruction in laryngeal tuberculous lesions of the open ulcerative type, but clinical research in this field has not yet been completed. In advanced cases, laryngeal tuberculosis may require tracheotomy because of laryngeal obstruction. The patient should not be allowed to struggle along with severe dyspnea even though not in danger of asphyxia. Distress and loss of sleep have an unfavorable effect on both the pulmonary and the laryngeal lesions. The tracheal incision should be below the third ring.

Prognosis.—The prognosis of laryngeal tuberculosis is closely interlocked with the prognosis of the accompanying pulmonary tuberculosis. The advent of the laryngeal lesion is regarded by all pathologists as adding seriously to the prognosis of the pulmonary disease, yet it is an unquestioned fact that if the pulmonary lesions heal the prognosis of the laryngeal tuberculosis is good. Extrinsic laryngeal lesions produce pain and odynophagia that interfere with the essential rest and nutrition. Means and willingness cheerfully to conform to a strict regimen in the most favorable environment render the prognosis good except in advanced stages. If the patient insists on talking all the time he is awake and similarly disobeys all other orders the prognosis is bad. Systemic syphilis worsens the prognosis, chiefly because of the unfavorable effect of the necessary antisyphilitic medication. Pregnancy is an unfavorable factor.

Sequelae.—Stenosis of the larynx is the chief sequela. As a rule it had better not be treated surgically lest it reactivate the pulmonary tuberculosis.¹

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LUPUS OF THE LARYNX

Lupus of the larynx is a clinical type of tuberculosis histologically indistinguishable from the common form of laryngeal tuberculosis.

Compared to the common type of laryngeal tuberculosis, lupus is rare but the cases are sufficiently numerous definitely to classify the type as a clinical morbid entity. Compared to this country, it is much more prevalent in Europe, especially in the northern sections where long, dark, foggy winters minimize sunlight.

Etiology.—The exciting cause is a bacillus identical with the *Mycobacterium tuberculosis*, so far as the bacteriologic science of today can determine. The disease is contagious and when transmitted the clinical features are maintained. The source of the laryngeal infection may be a lupoid lesion in the pharyngeal or nasal mucosa. It seems rarely if ever to come from a pulmonary lesion. Bacteriologically it has all the features justifying classification as an attenuated variety of specific infection. The *Mycobacterium tuberculosis bovis* has not been found in the laryngeal lesions but this may be due to the difficulty of finding bacilli in laryngeal tissue. The bovine organism has been demonstrated in the nasal and pharyngeal lesions. Poor living conditions, inadequate food, avitaminosis, insufficient sunlight, and dust laden atmosphere are important, one might say essential, predisposing factors. Anxiety and overwork may be contributory.

Morbid Anatomy.—Histologically the description of tuberculosis of the larynx fits the lesions of lupus. The lesions are usually more superficial and they show more fibrosis, but the differences are not usually sufficient for a pathologist to say from histologic evidence alone that a case is one of lupus of the larynx. Grossly the lesions show a characteristic tendency to cicatrize at one place while developing a new lesion at another. Though the lesions are rather superficial they cause stenosis requiring tracheotomy in some cases. One pathologic

peculiarity is that the thyroid and cricoid cartilages are not involved yet the epiglottis rarely escapes. The crico arytenoid joint also usually escapes. The vocal cords are less often involved as compared to the aryepiglottic folds and ventricular bands. Surface exudates are ad herent and patchy.

Associated Disease—In contrast with typical tuberculosis of the larynx, lupus is nearly always associated with a similar lesion in the pharynx, nasal cavities, or external nose. The latter condition is known as lupus vulgaris, as distinguished from lupus erythematosus which is not a tuberculosis. Another clinical contrast is that, whereas laryngeal tuberculosis is practically always secondary to pulmonary tuberculosis, lupus commonly occurs in patients with no demonstrable tuberculous lesion in the lung, and in a few cases pulmonary tuberculosis may follow lupus of the larynx, a reversed sequence as compared to typical laryngeal tuberculosis. The usually associated *lupus in the pharynx and on the palate* is a characteristic mucosal lesion. If recent, there will be tiny yellowish or pink nodules, 1 mm or more in diameter. At a later stage, similar lesions may be seen in one location while in another location the nodules have coalesced to form shallow ulcers partly covered with adherent secretion. In other locations, scarring may be visible. The lesions seem to stop short usually at the edge of the anterior pillar, only in relatively few cases is the palatine tonsil involved, though the velum may be shrunken to a mere fibrous remnant. The posterior surface of the velum may be affected as well as the anterior, but, curiously, perforation does not occur. All of these lesions except the scarring may disappear. Lesions of lupus or sequela scars are usually present in the nose.

Reflected Appearances—If the epiglottis is involved the characteristic appearances of an early lesion are scattered points of yellowish or pinkish small elevations on a pale mucous background. Later these points become more numerous and also join by peripheral extension, on coalescence they break down into a superficial ulcer with an irregular and undefined border. The ulcer may be coated with scanty, sticky secretion. If a patient is seen at a later stage there may be a scar where such an ulcer has healed, and along with the scar there may, or may not, be fresh dots of new lesions, or an ulcer in a new location. In a late case there may be seen a marginal irregular notching of the

epiglottis, or a mere stump of it remaining, ulcerated or deformed and cicatrized. The deeper lesions seen are pale infiltrations resembling edema in appearance involving the aryepiglottic folds and ventricular bands. The vocal cords are rarely affected, but when they are the appearances are those of tiny nodules, small erosions, or superficial ulcers.

Appearances on Direct Laryngoscopy.—On palpation with the closed laryngeal forceps, the edematous appearing aryepiglottic folds and ventricular bands are found to be moderately firm tissue, not a true edema. On lifting the normal ventricular band a lesion that was invisible may be revealed. The coous elasticus usually shows tiny nodules in its upper part when explored with the mouth of the tube.

Symptoms—One of the striking characteristics of the disease is its painlessness. The degree of ulceration in the extrinsic area that would cause constant pain and odynophagia in tuberculosis or cancer, may in lupus cause so little pain that the patient may complain only of stiffness in swallowing or of hoarseness which really comes from an endolaryngeal part of the lesion. This degree of anesthesia occurs in no other disease except leprosy. Cough and pulmonary symptoms are usually absent.

Diagnosis—The diagnostic peculiarities are (1) painlessness out of all proportion to the appearance and site of the lesion, (2) absence of odynophagia, (3) tendency to healing, (4) scarred areas associated with areas of erosion and ulceration, (5) absence (in most cases) of pulmonary and systemic symptoms, and (6) negative serologic reaction to the Wassermann test and absence of all signs of systemic syphilis. In a lesion or lesions of these characteristics, and of the appearances mentioned, a biopsy specimen showing the histologic changes of tuberculosis is significant. No harm whatever will follow nipping off of a few small nodules from the larynx for biopsy study, but often the specimen can be obtained from the accompanying pharyngeal or nasal lesion.

Treatment—Local treatment is not required except in the cases in which encroachment on the laryngeal airway may necessitate tracheotomy. The natural tendency of lupus to heal requires for cure only the aid of a regimen of silence, elimination of tobacco, avoidance of alcohol as a beverage and improvement of the living conditions and environment. Almost all these patients need sunshine. If a sunny en-

environment cannot be had, light treatment will be helpful. When available, sunlight reflected onto the lesion by a system of plane mirrors is good treatment. A concave mirror, like a head mirror, would cause a burn. Freedom from anxiety and overexertion are advisable, so are rest and fresh air, but in most cases these patients do not need the strict regimen of pulmonary tuberculosis except in the rare cases in which this disease occurs as a coincidence.

Prognosis—Restoration of function is the rule rather than the exception. Asphyxia has been known to occur but only for want of a tracheotomy that was long and clearly indicated. Even a coincidental pulmonary tuberculosis does not endanger life if proper care is taken. Recurrences are unlikely unless there is a return of the original predisposing factors.

Sequelae—Scarring remains but cicatricial stenosis is a rare sequela.

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ACUTE TUBERCULOSIS OF THE LARYNX

Acute tuberculosis of the larynx is tuberculosis of the larynx that runs an extremely rapid course. One clinical type may be properly called *acute milary tuberculosis of the larynx*.

Primary acute laryngeal tuberculosis and secondary acute laryngeal tuberculosis are not uncommon, but acute milary tuberculosis as a phase of general acute milary tuberculosis is relatively rare.

Etiology and Pathology.—Bacteriologically there are no demonstrable differences in *Mycobacterium tuberculosis* adequately to explain the different clinical types. It is a well known fact that virulence varies in all pathogenic bacteria. There is the equally important factor of variation of resistance not only as among individuals but in the same individual at different times. In some cases there may be an obvious cause for this, for example, pregnancy, starvation, great grief, change to unfavorable environment. Sudden access to the blood stream by a

large quantity of organisms from breaking through of an active lesion causes widely disseminated foci, one of which may be in the larynx. The breaking through of an active focus into the bronchus produces a valvular obstructive atelectasis causing an acute, and usually a quickly fatal, illness, but not a dissemination of foci, and it does not affect the larynx.

Varieties of the Disease—There are three clinical types of acute laryngeal tuberculosis. ① In a patient with a pulmonary tuberculosis but not a previously demonstrable laryngeal lesion, one or more nodules develop rapidly and break down into a deep ulcer that spreads rapidly. Soon similar lesions appear in other parts of the larynx. Within a few months, if the patient should live that long, the larynx goes through the stages of ulceration, true edema, perichondritis, chondral necrosis, stenosis, and impending asphyxia that, in a chronic case, might require a year or two. ② A patient with a chronic laryngeal and pulmonary tuberculosis takes a sudden turn for the worse, as though all resistance were suddenly lost, the terminal stages develop rapidly as in the preceding type. ③ In some cases there is an acute milary tuberculosis associated with a similar disease involving the lungs and other viscera.

Reflected and Direct Appearances—In types 1 and 2 the lesions are similar in appearance to those of chronic laryngeal tuberculosis, but if examined repeatedly at intervals the rapid progress will be obvious. In type 3 the milary tubercles are seen as small yellowish points on a semitranslucent background of swollen mucosa. Within a few weeks the little tubercles are seen to have coalesced into ragged ulcers that spread and deepen rapidly. The epiglottis and the aryepiglottic folds are the usual locations, and in almost all cases the pharynx is invaded by a similar process. The characteristic aggressiveness of the lesions is soon obvious.

Symptoms—The laryngeal symptoms are the same as those of chronic tuberculous disease, but of greater severity, as are also the general symptoms. In type 3 there are the profound general symptoms of acute milary tuberculosis similar to those of typhoid fever. Pain is more severe than in the chronic form of laryngeal tuberculosis.

Diagnosis—A few weeks of observation will reveal the rapid progress of the lesions, which is in definite contrast to the indolent course of

chronic laryngeal tuberculosis. If any doubt should exist as to the condition being tuberculous it can be promptly settled by histologic examination of a specimen taken by direct laryngoscopy. If as is usual there is a similar lesion in the pharynx the specimen may be taken from that location. The accompanying systemic symptoms afford additional evidence. As in all other cases a serologic test for syphilis should not be omitted.

Treatment—Palliative treatment as given for advanced stages of the chronic laryngeal tuberculosis is indicated. Systemic treatment according to indications is of utmost importance.

Prognosis—The prognosis is bad but only because of the lesions elsewhere and of the associated systemic conditions. The patients do not die of the laryngeal condition unless of asphyxia for lack of a tracheotomy.

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CHRONIC CICATRICAL STENOSIS OF THE LARYNX

A postinflammatory diminution of the lumen of the larynx sufficient to prevent decannulation is known as chronic cicatricial stenosis of the larynx. If the lumen is obliterated at any location it is a *total atresia*. Either condition may involve also the trachea forming a *laryngo-tracheal stenosis* or *laryngo-tracheal atresia*. *Arthritic fixation of the crico-arytenoid joint* is a form of cicatricial stenosis.

Prior to the demonstration of the then usual high tracheotomy as the cause of laryngeal stenosis, fully 50 per cent of children tracheotomized for diphtheria could not be decannulated because of stenosis. Tracheotomies on adults in various diseases were followed by about half this percentage of stenoses. Not more than 5 per cent of tracheotomies today present this sequela. Even this small percentage could be further reduced by the methods herein given.

Etiology—In the past the chief cause was high tracheotomy. A common occurrence was the arrival of a patient who originally had only a bilateral abductor paralysis to which a cicatricial stenosis was needlessly added by a high tracheotomy. The high operation is still done occasionally with resultant stenosis. Extensive loss of cartilage complicating perichondritis is a cause. Syphilis, diphtheria, typhoid fever, and trauma are frequent factors in stenosis. Oral sepsis and especially virulent infections, blood dyscrasias and malnutrition are contributory causes. Neglect of proper daily toilet of the cannula (*q. v.*) favors building up of cicatricial tissue above the fistula. Many patients referred to the clinic have arrived wearing a filthy corroded cannula that had not been changed for a month or more.

Pathology—In the larynx, as in all tubular anatomic structures, prolonged inflammation builds up small round-cell infiltration which becomes fibrotic and forms a stricture by persistent contraction. Necrotic loss of cartilaginous laryngeal framework impairs resistance to contraction. Necrosis of tracheal rings has a similar effect. Arthritis of the crico-arytenoid joint results in limitation of movement or rigid fixation, often bilateral.

Symptoms—Dyspnea and impaired voice are the chief symptoms. If tracheotomy has been done, dyspnea will be present only when the opening in the neck or cannula is occluded. The degree of vocal impairment ranges from slight hoarseness to a hissing huskiness or an apnoea. If any air passes through the larynx there will be at least a whispered voice. If a total atresia has persisted for a long time the patient may have a buccal pharyngeal or esophageal voice (*q. v.*). In cases in which tracheotomy has been postponed a long time a funnel breast may be present.

Diagnosis—Reflected appearances may be suggestive but direct laryngoscopic appearances are unmistakable to any examiner who is familiar with the appearances of the normal larynx.

Examination—The scar tissue by its persistent contraction distorts the interior of the larynx into one of many different forms. The lumen remaining may be of any shape from a slit in a more or less regular circle. All landmarks may be gone. On the other hand the larynx may look normal in the mirror, with a surprisingly good phonation yet the cords do

not separate on inspiration, and on examination with the direct laryngoscope the passive mobility test (*q v*) will show an arthritic fixation of the crico arytenoid joint. The cases in which the original obstruction for which the tracheotomy was done has disappeared (bilateral midline paralysis, for instance) but a cicatricial stricture persists, because of faulty tracheotomy, may be called those of *posttracheotomic stenosis*. There is occasionally encountered a *progressive subglottic fibrosis*, probably of chronic infective origin, though the history may include no record of any of the diseases that usually cause a cicatricial stenosis. If the patient arrives before a tracheotomy has been necessitated, direct laryngoscopic dilatational treatment may avoid tracheotomy. In any case of laryngeal stenosis the trachea may be involved—a condition called a *laryngotracheal stenosis*.

Complications—Persistence of the causative disease, such as syphilis, tuberculosis, and perichondritis, is a complication always to be borne in mind. Pulmonary complications are not infrequent, especially under inadvisable forms of treatment.

Prophylaxis—Early use of core molds, shortening of the healing period, and low tracheotomy, with daily toilet of the wound and proper technique of decannulation, are the best means of prevention. If a high incision of the trachea has been done by reason of stress of emergency, or by trauma, a cannula should not be worn in the high position. As soon as normal respiration has been restored a lower tracheotomy, with an interval of at least two uncut rings, should be immediately done for the wearing of a cannula, even if the prospects are good for a short duration of the acute stenosis that has necessitated the tracheotomy. The prevention of stenosis after injuries is considered under "Trauma of the Larynx" (p. 552). If, in any kind of case, stenosis cannot be prevented, total atresia can and should be prevented, core-mold treatment is the best method, but even a braided silk thread, worn "endless" (through mouth, larynx, and tracheotomic fistula) will maintain a fistulous lumen that can be subsequently dilated by core mold treatment, without the necessity of getting a start by perforation.

Indications and Contraindications for Treatment—If the patient cannot comfortably dispense with a cannula, treatment should be

advised, unless contraindicated by such preceding or present diseases as cancer or tuberculosis, or by a life expectancy so short from any cause as to make prolonged treatment not worth while.

Treatment—Patients with lesser degrees of cicatricial stenosis are usually curable by bi-weekly dilatations with the triangular dilator (Fig. 366). If the subglottic region or the trachea is involved the round dilator (Fig. 366) is better. Either is passed (lubricated with liquid petrolatum) through the stenosis, by means of the direct laryngoscope without anesthesia, general or local. Larocaine may, however, be used for the first few treatments if desired, as mentioned in connection with the technique of direct laryngoscopy.

If the patient arrives wearing a tracheotomic cannula the first step in the treatment is to determine the exact location of the opening in the wall of the trachea. This is done by insinuating

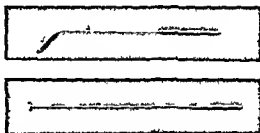


Fig. 366—Dilators for use through the direct laryngoscope in the treatment of cicatricial stenosis of the larynx and trachea and bronchi.

a small bronchoscope through the stenosed laryngeal lumen. If there is no lumen the determination is made by palpation of the cricoid cartilage and estimating the distance of the opening below it. If the old opening is not below the second ring of the trachea a new tracheotomy should be done, leaving, if possible, two good tracheal rings between the old opening and the new, otherwise the cannula will be constantly sliding up into the old opening. After about a week, treatment may be begun. During this week the patient should be thoroughly studied to determine the presence of any of the contraindications mentioned. If systemic syphilis is present treatment of the stenosis should be postponed until after the patient has had about a month of antisyphilitic treatment (p. 20). During the week of study, local endoscopic examination of the stenosis is used to determine the plan of treatment.

If the stenosed lumen is found to be so small as to cause distressing dyspnea, as determined by occluding the cannula with a whole cork (Fig 367), the dilatational treatments will not be likely to yield satisfactory results and the treatment by core molds should be begun at

stricted lumen. The greatest expansile pressure should be at the proper place, and this is taken care of by the shape of the core mold by reason of its form which closely approximates that of the normal lumen. The technic of exposure of the larynx to view by direct laryn-



Fig 367 —Corks for decannulation after tracheotomy and also after relief of cicatricial laryngeal stenosis. They are ground from pure rubber cord. Commercial corks are dangerously friable.

once. The selection of size is of utmost importance. The methods given in the section on trauma of the larynx refer to use of core molds in acute conditions (p 556). The chief difference in the treatment of cicatricial stenosis is that the core mold must be a tighter fit when introduced, in order to make enough elastic pressure

gосopy has been given on a previous page. As soon as the laryngeal orifice comes into view the axis of the lumen of stricture must be studied because the axis must be constantly in mind during the introduction, this is of utmost importance. The head of the patient must be raised or lowered a little so as to bring the axis

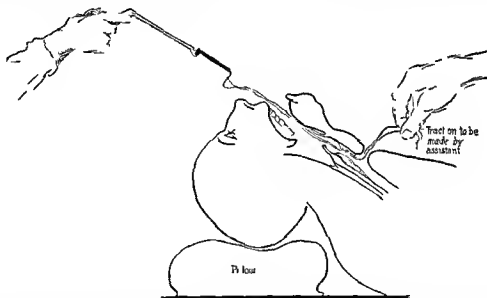


Fig 368 —Sketch illustrating the technic of placing a core mold for the treatment of cicatricial laryngeal stenosis. With the introducer the operator introduces and maintains the core mold in proper position with overhang backward, while the assistant makes traction from below to overcome resistance as the core mold enters the stricture indicated by the wavy line shading. The traction is rendered axial by the push fork (Figs 369-370). The proper position of the core mold is shown in Figure 371. The double braided silk (No. 14) shown here is pulled through with a thread passed with the instruments shown in Figure 372.

to cause absorption of cicatricial tissue and enlargement of lumen. This, however, does not mean that so large a size shall be used as would necessitate the use of undue force in introduction. In estimating the degree of resistance indicative of proper fit the operator must be certain the core mold is following the axis of the

to correspond with the operator's visual axis (Fig 342). The instrument nurse hands to the operator the introducer properly armed with the core mold, which is lubricated with sterile water. The operator, with a clear conception of the axis of the strictured lumen in mind, introduces the core mold and insinuates it into the

orifice of the lumen and pushes on it until the supraglottic swell rests in the aditus laryngis with the interarytenoid projection backward. The introducer is then released and the core

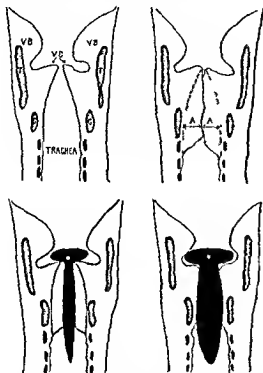


Fig 369—Above At the left is shown a schematic cross section of the normal larynx VB identifies the ventricular band VC, the vocal cords T, the thyroid cartilage C, the cricoid cartilage. At the right is shown a cross section of a larynx and trachea as affected by massive cicatricial hyperplasia AA indicates the hyperplastic tissue obliterating the lumen of the larynx below the level of the ventricular floor. It was especially firm and obliterative through the cricoid region.

Below At the left is shown a schematic cross section of the strictured larynx with a core mold of slender diameter. The tissue AA was so firm and filled the cricoid ring so completely that it prompted a proposal to cut the cricoid cartilage. At the right is represented a core mold of large size in position after progressively increasing sizes of molds had caused absorption of the hyperplastic mass (AA at right above). Disastrous cutting of the cricoid cartilage was shown to be unnecessary. This mold was of special shape.

(NOTE—All our clinical experience has been with core molds ground to shape out of pure rubber cord made from natural rubber. Cautioning against the use of any of the various forms of synthetic rubber seems advisable, no one knows what the reaction of the laryngeal tissues to them might be. Synthetic rubber is a physical not a chemical imitation.)

mold is left in situ by opening the ring handle of the introducer. The operator looks at the core mold to assure himself that the supraglottic swell remains in proper position in the vestibule of the larynx.

Until facility is acquired there are certain difficulties and pitfalls. The best way to avoid most of these is to practice the motions hundreds of times on the cadaver, or on a large rag-doll manikin such as any nurse can make. Like most manual things knowing how is not enough, manual training is necessary. The core mold must be lubricated with water, never with oil or grease. To make the water spread evenly over the surface, the mold should be washed with soap and water and sterilized in a grease-free sterilizer. It is then put in a small pan of sterile water on the instrument table. The instrument nurse must be instructed to put the core mold on the introducer with the overhang of the supraglottic swell downward, which will be



Fig 370—When the introduction of a slender core mold meets with much resistance, it may be assisted by traction from below. In order to make the traction axial, the push fork depresses the braided silk (B) into the trachea when traction is made. To prevent any possibility of plunging of the fork into the tissues, the operator grasps the push fork tightly and rests the ball of the thumb, DD, on the front of the patient's neck, H. As landmarks, the epiglottis, E, and the arytenoid (A) and cricoid (C) cartilages are represented. The hand is sketched ungloved for clearness.

posterior in the recumbent patient. If the core mold will not enter, the operator must make sure it is properly directed before resorting to a size smaller. In such a case it is better to use the traction method of seating the core mold as in cases of total atresia, described in a subsequent paragraph (Figs 368, 369, 370). In fact it is well in any case to have the aid of traction rather than to depend upon pulsion alone. The laryngoscope may be dispensed with but the introducer is indispensable to prevent the core mold from rotating. The interarytenoid overhang must be backward.

When, for lack of an endless thread or other means of maintaining some kind of a fistulous track through the larynx, a total atresia of the larynx has formed, a perforation must be made to get a start with the smallest core mold. Thus

is done with the perforating instrument shown in Figure 373. Before it is used, however, the transilluminators (Fig. 374) must be used to determine the thickness of the intervening ob-

be made, the perforator with the sheathed blade pointing anteriorly is introduced and the perforation made. The braided silk in the eye of the perforator is caught with forceps by the

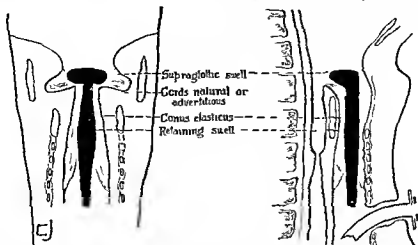


Fig. 371—Sketches of sagittal and coronal cross section of the neck illustrating schematically the fundamental principles of the core mold: its action and its retention. The supraglottic swell molds the ventricles and prevents descent of the core mold. The infraglottic swell molds the conus elasticus and prevents expulsion of the mold by cough. The overhang of the supraglottic swell is backward between the arytenoid eminences. These sketches represent a midway stage, the molding will be complete when all the cicatricial tissue represented by wavy line shading, becomes absorbed from the long-continued elastic pressure of progressively increased sizes of core molds.

structive tissue and the direction the perforation should take. The light is passed up from the tracheotomic fistula, by an assistant who carefully maintains the transilluminator in the axis of the trachea. The operator looking down the

assistant who held the transilluminator, the loop of silk is drawn out the tracheotomic fistula and held while the operator withdraws the perforator. This leaves a double silk thread

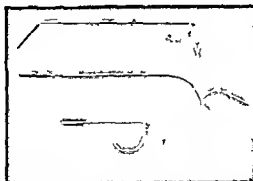


Fig. 372—Above: Silk carriers for passing braided silk upward or downward through the mouth, the cicatricially stenosed larynx, and out through the tracheotomic fistula in the neck. Below: Bodkin for threading the carriers, also for threading core molds, perforators or other instruments.

laryngoscope sees the spot of transillumination. The spot will be easier seen if the light on the laryngoscope is dimmed by partial withdrawal of its light carrier, but this is not necessary unless the intervening tissue is thick. Having determined the spot at which perforation is to



Fig. 373—Sheathed knife perforator to be used to carry silk through the scar tissue in cases of total obliteration of the lumen of the larynx.

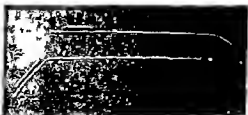


Fig. 374—Transillumination for locating the thinnest and best place for perforating the scar tissue in cases of total obliteration of the laryngeal lumen.

running through the mouth, stricture, and tracheotomic fistula, with which to pull down on the lower end of the core mold (Figs. 368, 369, 370, 371). It must be clearly understood

that this traction does not dispense with the use of the intubator. The intubator is needed to push on the core mold synchronously with the traction from below, and it is also absolutely

projects low enough to obstruct the tracheotomic fistula, the core mold may be shortened by cutting off enough of the core mold and grinding the cut edge smooth on an emery

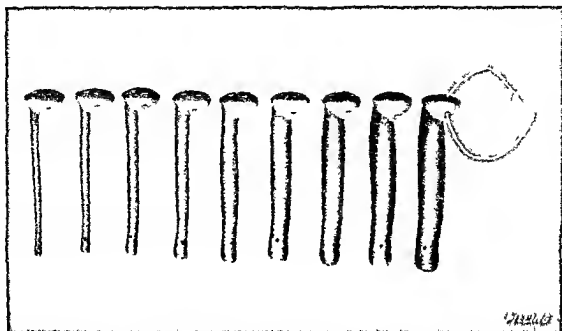


Fig. 375—Core molds of nine standard sizes and shapes. These are adult length; they can be shortened by grinding off to suit the measurements of children. Special shapes can be made to suit particular cases, such as for example the case illustrated in Figures 369 and 370.

essential for preventing rotation of the core mold, thus insuring the posterior placement of the overhang of the supraglottic swell (Fig. 371). One last point is vital, namely, the lower end of the core mold must never project below the tracheotomic fistula lest it asphyxiate the pa-

wheel (a sand paper wheel is better, a manicurist's emery board will do). If, however, the lower end of the core mold is needed for dilatation of a tracheal stenosis close to the fistula a lower tracheotomy must be done and the commencement of the core mold treatment delayed

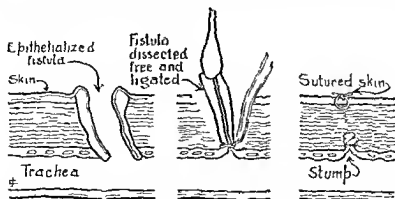


Fig. 376—A simple plastic procedure for closure of an epithelialized fistula that sometimes persists after prolonged wearing of a tracheal cannula. The fistula wall is dissected free and excised after ligation. The stump is touched with carbolic acid and the skin is closed over it.³

tient in case the cannula should accidentally come out, or be removed by the patient, in the absence of a physician or a nurse familiar with the apparatus. If the lower end of the core mold

for two weeks for the formation of a new and low fistula.

When a core mold is properly placed, the tracheotomic cannula is inserted and tied with

its tapes. An efficient core mold will loosen itself so that it would be expelled spontaneously in a few weeks. To prevent spontaneous expulsion the two ends of the traction silk strings are tied together in a double bow knot on the curve of the tracheotomic cannula. This permits the untying and retying at the *daily toilet of the cannula* (q v). When the core mold becomes a loose fit, as determined by palpation with the closed laryngeal forceps used through the direct laryngoscope, it is removed by grasping it with the introducer in the same way as when introducing. The anchoring braided silk is tied again over the cannula. This is repeated with each size up to a little over the normal luminal diameter of the larynx, about 38 F in an adult (Fig 375). This size is usually worn, with biweekly replacements, for three or four months. Then a test is made to be certain a cure has been obtained. This is done by leaving out the core mold while keeping the patient in the hospital under close observation. Until the test is completed, the tracheotomy fistula must be kept from healing (that is, if there is a tendency to heal) by using a silver plug if necessary.

Tracheal Stenosis—If the tracheal stricture is part of a laryngotracheal stenosis all that is required for cure is treatment with core molds long enough to include the tracheal stricture, the cannula being suitably lowered. Cicatricial stenosis involving only the trachea yields readily to biweekly dilatations with the round dilators (Fig 366) used through the direct laryngoscope. *Thymic compression stenosis of the trachea* requires only roentgen ray therapy. Other forms of compression stenosis, as by a tumor, may require the cane shaped cannula, to be abandoned later if and when the tumoral compression is relieved. If "rings" are gone, new "rings" of cartilage to hold the trachea patulous can be constructed out of cartilage from the external ear.³ The so-called rings of the trachea are naturally incomplete and somewhat resemble a horseshoe in shape.

Plastic Closure of a Tracheotomic Fistula—If a cannula is worn for only a few weeks the fistula closes promptly after removal of the cannula. If, however, a cannula has been worn for months the fistula will have become epithelialized and this epithelial lining prevents

healing. There is little air leakage but a little moisture on the clothing from leakage of mucus is annoying to the patient. The fistula can be closed and only an almost invisible scar will remain after the simple plastic procedure shown in Figure 376.

Prognosis—If the condition is purely cicatricial most of the patients can be cured in a few months. If there has been loss of laryngeal framework the period of treatment required will be prolonged proportionately to the amount of the loss. In general it may be stated that the ultimate prognosis is good both as to function and life under treatment. The use of core mold methods has rendered obsolete the old idea of trying to reconcile the patient to wearing a cannula for life, an idea that often led to dread and dangerous postponement of tracheotomy. Many children whose laryngeal stenosis had been pronounced hopeless have been ultimately and permanently cured. In these extremely difficult cases, in which there is only a shapeless remnant of cartilage out of which to make a framework with a lumen, continuous and prolonged patient work during the growing period is essential.

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NONINFLAMMATORY STENOSIS OF THE LARYNX

Noninflammatory stenosis of the larynx designates laryngeal obstruction of other than inflammatory origin. The term applies only to the primary condition, any primarily noninflammatory laryngeal lesion may be and often is followed by secondary inflammatory processes. Various synonyms apply to particular conditions. There is much improper use of the noun "laryngitis," it is commonly applied to many conditions that are not inflammatory hence do not justify use of the suffix "-itis." Some forms of trauma and paralysis are primarily noninflammatory. Some anomalies are obstructive. Noninflammatory edema of the larynx is not uncommonly associated with myxedema, cretinism,¹ nephritis, cirrhosis of the liver, or cardiac or vascular diseases. The diagnosis and treatment are general medical problems. Multiple puncture of the laryngeal edema, often advised, is a mistake. A noninflammatory lymphedema of the larynx sometimes follows long after roentgen-ray therapy.

Angioneurotic Edema of the Larynx.—This rather uncommon disease is caused by the same factors as the dermal giant urticaria (Quincke's disease) but it occurs, in some instances, as an independent or an alternate manifestation. Known basic factors are heredity and the type of individual subject to functional disturbances of the nervous system. Allergy is a factor in some cases. *Pathologically*, angioneurotic edema of the larynx is one of the little understood vegetative vasomotor disorders. The vessels are dilated and the perivascular tissues of the mucosa and submucosa are distended with clear serum, even the cells may be edematous.² The reflected appearances usually are of three rather purplish mounds with no landmarks discernible. The anterior mound represents the epiglottis, the lateral mounds are each made up of the aryepiglottic fold and the ventricular band fused together, the ventricle being obliterated. This is the appearance at the stage of maximum development. At an earlier stage the structures mentioned are pinkish in color and swollen in form, and below each cord on each side is visible a smooth, bulging, pinkish swelling of the tissues of the conus elasticus. This early stage may last only a few minutes, we have seen it develop to the maximum under observation with the mirror.¹ The symptoms are

a tickling sensation quickly followed by dyspnea and the cardinal signs of obstructive laryngeal dyspnea ($q \vee$). The voice is muffled, guttural, and deep in pitch.¹ The diagnosis is readily made by the reflected appearances, the suddenness of the laryngeal obstruction, and the associated dermal and urinary symptoms. Though the laryngeal lesion may occur alone there is almost always a history of previous occurrence of the associated symptoms. General examination may show a residual chronic edema, on the lip for example. *Prophylaxis* is effective when allergy is an exciting factor. Prevention of asphyxia requires that the patient remain always in reach of prompt surgical relief. General medical care and management will help in preventing recurring attacks, or lessening their frequency. *Treatment* of the larynx by inhalation of a spray of ephedrine 1 per cent in isotonic salt solution, or inhalation of benzedrine vapor may be tried, but anything irritating will do more harm than good. Puncture would be a serious mistake. As a rule the edema subsides spontaneously in a few days. However, tracheotomy is necessary in most cases to prevent asphyxia during the maximum stage. If asphyxia be prevented there is little or no danger to life and the prognosis for recovery of all nine laryngeal functions is good. A single attack usually leaves no trace. Following repeated attacks there may be a permanent diffused swelling that gives the larynx a "gingerbread" appearance but is not seriously obstructive. In one of our cases, a specimen taken to exclude tuberculosis showed the post-angioneurotic condition to be only an increase in the connective tissue.

Lymphedema of the Larynx.—This is a thickening of the endolaryngeal tissues due to abnormal conditions in the lymphatics. As a primary local condition it is rare, secondary to irradiation it is common, though with one exception it is not referred to in the textbooks.¹ The fundamental morbid anatomic condition is stasis of lymph. This may be due to local or distal obstruction in lymph channels or nodes. Angioneurotic edema ($q \vee$), if it occurs repeatedly and at short intervals, is followed by a chronic lymphedema in at least half of the individuals thus affected. A single attack rarely has this sequela. Tuberculosis is a rare cause, it may produce lymph stasis by a laryngeal, cervical, or intrathoracic tuberculous lesion, or by surgical procedures, for example, removal of

tuberculous nodes. Once a lymph stasis becomes established there is created a vicious circle. The distention of the lymph channels renders their valvular mechanism inefficient, this creates more stasis by increasing pressure on the channel walls, increase in degree and area of dilatation follows. Collateral circulation of lymph channels is quickly established but the collateral channels in an area may also be obliterated. Another factor in this vicious circle is fibrosis. Naturally the increase of fibroblasts favors fibrosis and the increased fibrosis increases obstruction and lymph stasis. This vicious circle is often the result of irradiation by the roentgen ray and by radium. It is not a part of the acute reaction of irradiation that appears within a week or two of the exposure. It develops many months after the causative irradiation, often long after a cure by irradiation, and has the appearance of a primary disease. In some cases this form of lymphedema is apparent only in the larynx, but we have seen it also in the esophagus, usually, however, it is apparent first low in the neck and gradually rises until the larynx is reached. Lymphedema of the larynx from irradiation, is, of course, an entirely different thing from the serous edema resulting from either the mucosal reaction or the perichondritic and chondral necrotic sequelae of irradiation.

As seen in the mirror the color of the mucosa is usually paler than normal. In form the characteristic appearance in a well developed laryngeal lymphedema is a diffused smooth rounding of all endolaryngeal structures with more or less obliteration of landmarks, giving a 'ginger bread' appearance. There is usually no apparent asymmetry if there is no laryngeal lesion other than the lymphedema. The appearances on direct laryngoscopy are distinctive. In a fully developed case of laryngeal lymphedema the outward displacement of the ventricular bands at the approach of the anterior commissure laryngoscope reveals an almost complete filling of the ventricle. The cords are seen to be less edematous than other parts of the larynx. On palpation, with the closed laryngeal forceps used as a probe, the edema is found to be much firmer than an inflammatory edema.

The symptoms at first are chiefly vocal. As the lymphedema increases there develops a lack of clearness of tone and a lowering of the pitch of the voice, both of which changes slowly be-

come more and more noticeable. Dyspnea may be slight even on exertion, if there is no other lesion or complication. In a few cases it eventually becomes extreme in degree, but the development is so slow the patient learns to get the maximum of air through a small glottic chink. As with any condition of diminished area of cross section of glottic airway, only a little intercurrent swelling may precipitate asphyxia. Progress is slow. Stenosis may become so great as to necessitate tracheotomy. Permanent gross thickening may impair the quality of the voice. Lymphedema of the pharynx may be associated in cases in which irradiation is the cause.

The diagnosis is made by the absence of inflammation which is so obvious a feature of the acute inflammatory edema appearing within a few weeks after operation. In this respect the reflected and direct appearances are characteristic but tuberculosis should always be excluded. Even in case of pulmonary tuberculosis, a lymphedema of the larynx may not be a tuberculous lesion. Biopsy will differentiate if the specimen be adequate and taken with the precision of direct laryngoscopic procedure.

Prophylaxis is difficult. In cases in which laryngeal edema might arise from a curable lesion in the neck or upper thoracic aperture, prompt attention to such a lesion might be preventive. In case of irradiation for malignant disease, obliteration of lymph channels is one of the objectives and subsequent lymphedema is a relatively minor and usually unavoidable matter. Treatment is disappointing. Tracheotomy may be necessary to prevent asphyxia. Removal of the cause is possible, and if known, is advisable though it cannot restore destroyed lymph channels if the channels of collateral circulation have also been destroyed. Considered apart from associated disease the prognosis is good as to life, but the edema does not disappear and the voice usually remains impaired.

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CONGENITAL ABNORMALITIES OF THE LARYNX

Structural or other abnormalities of the larynx present at birth may or may not be discovered until later, depending upon whether the larynx happens to be examined or not. The term *congenital laryngeal stridor* was applied to such cases in which this symptom was manifest in the days when diagnosis of infantile laryngeal disease was made by inference and theory.

There are no data upon which to determine incidence because it is only recently that use has been made of direct laryngoscopy as an easy means of examination of the infant larynx.

Etiology.—The protected anatomic position and the elastic suspension of the larynx justify eliminating from consideration, largely if not entirely, the secondary causative factors that are regarded as so important in the causation of congenital deformities of the extremities. Of the primary causes heredity and reversion may be factors. Shortcoming in the fetal power to metabolize calcium and phosphorus doubtless is a factor in some instances, but among the rather large number of affected young children who have come to our clinic there has been much evidence to show that the chief causes were hardships of which the respective mothers had been the victims during pregnancy, especially lack of sunlight, overwork, deficiency of diet in balance or quantity, avitaminosis, physical abuse, mental anxieties, and excessive childbearing. In some of the younger mothers there were evidences of osteomalacia. Prematurity at birth was apparently a factor in some of the children. Many of the fathers were addicted to alcoholic beverages. Whether this affected the fetus directly or indirectly through abuse and malnutrition of the mother is, of course, uncertain, possibly both means were factors. All of these observations were made in cases of congenital laryngeal abnormalities in children of the poor in Pittsburgh and Philadelphia.¹ The ages of the children at the time of observation ranged from birth to twenty-three months. The clinical facts justify the inference that the important factors in the causation of congenital laryngeal abnormalities are the same as those of fetal rickets (achondroplasia). In a few cases syphilis and accidents in accouchement were factors.

Pathology.—Anomalies, deformities, and other abnormalities may be in the soft tissues

or the cartilage or in both. Lack of normal stiffness is the most frequently found cartilaginous abnormality. Irregularity, asymmetry, and localized overgrowth are next in frequency, not always to a degree much beyond normal limits. Perichondritis was observed in a few among our series of patients, but in only one was it known to be present at birth. On histologic examination the cyst wall and the papillomas recorded did not differ from the same conditions occurring in older children.¹

Varieties of Congenital Abnormalities of the Larynx.—Objective examination of the larynx in the newborn and in the early life of children has yielded information on the subject of early abnormalities of the larynx that was unobtainable prior to the advent of direct laryngoscopy. The following are the more important conditions recorded.¹ *Chondromalacia* manifesting flaccidity instead of the normal resilience of the cartilaginous rim of the laryngeal orifice was found to be the most frequent of all the congenital abnormalities of the larynx. The margins of the epiglottis were seen to roll toward the midline on a vertical axis as the flaccid folds were drawn in over the laryngeal orifice by the current of air at each inspiration causing a stridorous sound.¹ *Hyperplasia of the epiglottis* was found to be rather a common abnormality. The overgrowth was seldom symmetrical and the cartilage was flaccid. As a result, the flapping at each inspiratory ingress of air caused a stridorous sound. *Hyperplasia of the ventricular bands* was recorded as one of the rarer forms of congenital laryngeal abnormality causing stridor. These bands are usually inconspicuous at birth. In one case stridor was noted on expiration. Expiratory stridor was noted also in a case of *supernumerary vocal band* and in one case of *congenital web*. *Total atresia* of the larynx was noted in one stillborn infant. *Pseudosyrinx*, a narrowing of the trachea leaving only a slitlike chink for passage of air, caused stridor on both inspiration and expiration. *Tracheal compression*, by congenital goiter, produced a similar condition in a few cases and thymic hypertrophy in a number. Both of these conditions are possible causes of obstructive laryngeal paralysis. Conspicuous among the recorded conditions that are classifiable as pathologic rather than anomalous were cysts, papillomas, laryngeal paralysees, myxedema, and dislo-

cation at the crico arytenoid joint and fixation at this joint ¹

Symptoms—Dyspnea and stridor are the chief symptoms, there may be also cyanosis or ashy grayness of the skin. Stridor is often present without dyspnea, especially in the less obstructive conditions. *Congenital laryngeal stridor* is the symptom common to all cases of noisy breathing in the newborn. At one time it was thought to be due to laryngeal spasm, but this was not observed in any of the cases to which reference has just been made. The stridor is usually inspiratory but in some cases it is expiratory, in other cases it is both. In the greater degrees of obstruction the cardinal signs of obstructive laryngeal dyspnea (q.v.) are present.

Diagnosis—There is only one way to make a diagnosis of congenital laryngeal abnormality during infancy and early childhood and that is by endoscopic examination. The objective discoveries of direct laryngoscopy have been a revelation in this field of laryngology ¹. The technique is elsewhere herein given.

Complications—Pulmonary atelectasis from tracheobronchial obstruction by secretions that the cilia, or cough, or tussive squeeze cannot expel is more likely to occur than pneumonia, though the latter is the erroneous diagnosis usually made in cases of laryngeal obstruction. Bronchoscopic or catheter aspiration is promptly curative in cases of obstructive pulmonary atelectasis of this type (Figs 358, 470).

Treatment—The treatment varies with the conditions found on direct laryngoscopy. If the obstruction is great a bronchoscope should be inserted for temporary relief. A gentle flow of oxygen and carbon dioxide (7 per cent) should be maintained by attaching the rubber tube to the side inlet of the bronchoscope. The precautions given under *Asphyxia Neonatorum* should be taken. As soon as respiratory regularity is established and the color of the skin is normal a low tracheotomy should be done. In cases in which there is only noisy breathing (stridor) without indrawing or signs of anoxemia, nothing need be done for anomalies. Between these two extremes there are many cases that require only intratracheal insufflation of oxygen and carbon dioxide (q.v.). An elongate epiglottis might possibly require amputation, but unless it is sufficient to endanger life by asphyxia it should be given a chance for spontaneous adjustment. It is probably always a

reversion Papillomas, cysts, or other tumors should be removed, and usually removal had better not be delayed. Medical care and management is essential in all cases, and in nearly all an antirachitic regimen should be followed whether definite evidence of rickets is present or not. This would, of course, include sunbath, skylight, cod liver and halibut liver oils, viosterol, or other source of vitamin D, also other vitamins, as deemed best by the practitioner in the particular case. Evidence of syphilis should always be sought and, of course, treated if found. The treatment of syphilitic children is given elsewhere herein. Thymic compression stenosis of the trachea is considered in connection with bronchial obstruction.

Prognosis—If asphyxia be prevented the prognosis as to both life and function is good. Every laryngologist occasionally sees in an adult a remnant of a previously undiscovered congenital abnormality, such as a web

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LARYNGOPTOSIS

Laryngoptosis is the name given to an abnormally low position of the larynx. Extreme degrees of ptosis are relatively rare but lesser degrees are common. The cause of the congenital form is unknown. Normally the larynx descends progressively from fetal life to maturity. Perhaps laryngoptosis in some cases is an excess in the rate and extent of this progress. We have seen congenital cases, however. In acquired cases the larynx is pulled down by adhesions, and by massive scarring of the neck. Any lesion causing displacement of the thoracic trachea will pull downward on the larynx, and this could be enough to cause a moderate ptosis, the cooperation of a favorable anatomic condition would be required for it alone to cause a high degree of ptosis. The only symptom is a monotonous voice with lowered pitch. Palpation renders diagnosis easy. If not more than

two rings of the trachea are above the upper border of the manubrium there is ptosis. If the thyroid or only the cricoid cartilage is below the sternum the notch of the thyroid can be felt in or above the guttural fossa. Laryngoptosis may complicate tracheotomy by compelling the operator to go through or above the larynx. Intrathoracic lesions may complicate laryngoptosis. Ordinarily no treatment is required for laryngoptosis itself, but associated conditions may require intervention.

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DYSPHONIA PLICAE VENTRICULARIS

Dysphonia plicae ventricularis means phonation with the ventricular bands. It may be either anomalous or vicarious.¹

The condition is not uncommon but is usually overlooked and statistically unrecorded. In one clinic where records of laryngeal disease were kept over many years it was found to be the cause in 4 per cent of patients complaining of hoarseness.¹

Etiology and Pathology.—There are two distinct types of case from the etiologic viewpoint: (1) the usurpative, in which the bands aggressively take over the phonatory function before the perfectly good cords have time to act, and (2) the vicarious, in which the bands try to phonate because the cords are absent, hindered, or imperfect. In the cases of the usurpative type there is often a hypertrophy of the overactive ventricular bands. The vicarious type is seen at its best in cases in which fixation of the crico-arytenoid joints or a tumor between the cords prevents proper cordal approximation on phonatory attempts. The constant effort of the patient to improve tone forces all the laryngeal muscles to utmost effort, so that the increasing activity and excursion naturally progress until ultimately the edges can touch and cut the air

column into the puffs that form tone. This development has been observed in its successive stages.¹ There is squamous epithelium over the edges of the ventricular bands, similar to that of the vocal cords, which probably favors development of vibratory action of the bands, when called upon to develop this function. As mentioned in connection with the physiology of the larynx the ventricles and ventricular bands normally cooperate in the phonatory function of the larynx, but the cooperation is subordinate.

Reflected Appearances.—In the usurpative form nothing is noted amiss on inspiration or quiet expiration. On phonation the ventricular bands are seen to close in advance of the vocal cords which are hidden by the closure of the bands above them. When the median edges of the bands meet in the midline they vibrate and a tone is produced. The cords reappear as the bands move outward on the following inspiration, and they follow along with this abductive movement. The impression given is that the cords could have been seen to vibrate if they had not been hidden from view by the prior closure of the bands above them. They probably do not vibrate in most cases because the crowding of the respective superjacent ventricular band acts as a damper in checking vibration. In some cases it is seen that the cords approximate and start to vibrate but are almost immediately overridden by the approximation of the bands which have taken over the phonation. It starts with a clear tone but almost immediately it becomes blurred and deeper in pitch. This produces a peculiar break in the voice that is annoying to some patients. It was noticed as a sound long ago and called *diphthongia* or *diplophonia* or double voice, but its true cause was only recently discovered. Once the possibility of phonation with the ventricular bands is realized as a diagnostic possibility it is readily recognizable, but it seems to be often overlooked, judging by the number of cases in which other diagnoses were made. The usurpative form is associated with other diseases that prevent approximation or vibration of the true cords. When the causative condition is a fixation of the cords in a separated position the arytenoid eminences seem to move inward a little as the ventricular bands approximate, but the cords remain fixed and disappear as the bands close over them. There is usually more or less of a gap between the approximated edges

Diagnosis—The first diagnostic step in every case of laryngeal disease is a serologic test for systemic syphilis whether this disease is suspected or not. If the reaction should be reported as positive plus 1 it should be repeated. If plus 2 or more further laryngeal methods of examination should be postponed until the patient has had antisyphilitic treatment. A therapeutic test should not be done unless the diagnosis of systemic syphilis is reasonably certain because it might befog the diagnosis. When the diagnosis of systemic syphilis is definitely made, it still remains to determine whether or not the laryngeal lesion or disability is syphilitic. Here is the proper sphere for the therapeutic test, and it should be in the form of energetic antisyphilitic treatment, omitting, however, the use of potassium iodide unless tracheotomy has been done. The tendency of this drug is to precipitate laryngeal edema. If the laryngeal lesion improves and the improvement is continuous the lesion is syphilitic. If the improvement ceases, biopsy should be done because even cancer of the larynx may be favorably affected for a time. As among the laryngeal lesions of cancer, tuberculosis, syphilis, and the mycoses, differential diagnosis is definitely made by biopsy, and the taking of a specimen for this purpose should be done without hesitation or long delay. The technic is given under "Cancer of the Larynx." A complete systemic and systematic examination is called for in all cases of laryngeal disease as elsewhere herein mentioned. In the diagnostic consideration of the larynx in systemic syphilis it must be borne in mind that laryngeal symptoms may be produced by a syphilitic lesion located in the thorax, the neck, or the cranium as well as in the larynx itself (see under Paralysis of the Larynx", also under 'Innervation of the Larynx")¹

Prophylaxis—The prophylaxis of systemic infection is important but is not within the scope of this textbook. The prevention of damage to the larynx by syphilis is of utmost importance because it is potentially possible in every case. It is another example of the wisdom of examination of the larynx in every case of any disease. The larynx of every syphilitic should be periodically examined. Additionally prompt re-examination should be made if the slightest hoarseness should appear. Even if the members of the family are not aware of the systemic infection a reliable member can be

told of the necessity for prompt laryngeal examination if any lack of clearness of voice should be detected. In most cases the beginning of syphilis of the larynx is overlooked under a homemade diagnosis of "laryngitis," and it is usually attributed to "catching cold." Efficient treatment of systemic syphilis will prevent the development of damaging laryngeal lesions. The prompt bacteriostatic action of penicillin given intravenously is invaluable in arresting potentially destructive lesions of late syphilis involving the larynx. Every patient, if not every human being, should have a serologic test.¹

Treatment—There are three phases of the problem of treatment: specific, supplemental, and local. First and most important is the specific treatment of the systemic disease. This is given in connection with syphilis of the nose (p. 20). The supplemental treatment consists of two important routines: (1) an excess of vitamins taken daily as a medicament as well as in the well regulated dietary regimen, (2) maintenance of a slight but continuous increase in elimination by bowels, skin, and kidneys. Local treatment is of lesser importance except for two things, namely, prevention of asphyxia and prevention of total atresia. If the cardinal signs of obstructive laryngeal dyspnea appear tracheotomy should be done without delay. Postponement may be justifiable when the patient is under the constant watchfulness of day and night nurses experienced in dyspneic cases and if the physician is promptly accessible, but it is axiomatic that tracheotomy is almost always done later than it should be done. All tracheotomies should be done low because high tracheotomy is the chief cause of chronic laryngeal stenosis,² but in no case is high tracheotomy more disastrous than in syphilis of the larynx. In these cases the tracheotomy should be done as far below the cricoid cartilage as the patient's anatomy will permit, below the third ring if possible. Endolaryngeal treatment of laryngeal syphilis is seldom advisable. If chemotherapy (q.v.) is deemed helpful for mixed pyogenic infections it had better be used orally or parenterally rather than locally. Puncture of edematous lesions is particularly to be avoided. After cicatrization has begun by reason of the systemic treatment, precautions against total atresia of the laryngeal lumen must be kept in mind. As a makeshift a heavy braided silk string worn "endless" through the nose, naso-pharynx, larynx, and tracheotomic fistula, may

be used, but the best way is with the smallest diameter of core mold. This is used only for prevention until cicatrization is nearly complete. After that, systematic treatment with core molds (*q v*) may be begun.¹

Sequelae.—Cicatricial stenosis will follow in most cases of ulcerative laryngeal syphilis if not prevented. Syphilitic fibrosis as distinct from cicatrization may result without ulceration. Both are amenable to core-mold treatment. Cicatricial syphilitic web may require excision in addition to the core mold method, but as a rule the elastic pressure of the molds will accomplish absorption of cicatricial tissue with better results without either incision or excision.¹

Prognosis.—Early secondary syphilitic laryngitis disappears spontaneously in most cases whether systemic treatment is given or not. Ulcerative syphilis has little or no tendency to spontaneous recovery and if untreated is disastrously destructive, it is amenable to systemic treatment. Fibrotic laryngeal syphilis is a parasymphilitic lesion that does not yield to systemic treatment, but its resultant stenosis will yield to core-mold methods. In general it may be stated that laryngeal syphilis is curable, in the limited sense of the word "cure" as applied to this disease. The only laryngeal danger to life is from asphyxia. The voice is usually impaired, but some kind of voice always remains if total atresia be prevented or cured.¹

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SCLEROMA

Scleroma is a chronic specific infective disease of the mucosa of the nose, pharynx, larynx, and trachea. *Scleroma respiratorium* is a synonym. *Rhinoscleroma* is used synonymously, but cases occur in which the nose is not affected.

The disease is common in Poland and becoming so in neighboring countries, especially Russia, Austria, Hungary, Rumania, Bulgaria, and Germany. A few cases have been reported from Italy, India, Egypt, and South America but the disease cannot be said to have established itself in these countries. At the Second International Congress of Otolaryngology at Madrid the Rapport of S. Beinoff included a collective investigation of Chevalier Jackson reporting forty-seven cases of scleroma in North America, of which forty-five were observed in the United States.¹

Etiology.—Scleroma has all the clinical features of an infectious disease. Though unanimity of opinion is lacking it is generally accepted that the causative organism is the *Bacillus rhinoscleromatis*. Probably no similar disease shows such colonies of these organisms embedded in the tissues. The geographic distribution of the disease, its total absence in many similar climates, seems indicative of an as yet unknown predisposing causative factor. To what extent the predisposition is climatic, geographic, or racial remains controversial. Unquestionably living conditions are an important factor, over 90 per cent of the cases occur under poor living conditions. Of forty-five cases occurring in the United States, collected by Chevalier Jackson,¹ seven of the patients were born in this country. One of our patients, the mother of a large family, has improved as to her general health and her scleroma during twenty years residence in this country, and no member of her family has contracted the disease.

Pathology.—The tissue changes are limited to the mucosa and submucosa. The apparent involvement of the skin of the upper lip is really a subdermal extension of the submucosal infiltration beneath the anterior nares and upper lip. The bulky, hard, infiltrated tissue, that seems almost cartilaginous to the touch, is fibrotic in character with no tendency to ulcerate. Embedded in this tissue are scattered, hyaline bodies, resembling droplets of oil, and numerous stain-resisting, usually nucleated, swollen, dropsical "foam" cells. Embedded in the tissue are the groups of encapsulated, gram-negative bacilli of Frisch, resembling those of Friedländer. The bacilli are most numerous in the foam cells, and these cells are most numerous in old lesions. The infiltration is bulky but may contract after a number of years. Any one or more of the following regions may be in-

volved primarily, secondarily, or as an extension by continuity anterior nares, under surface of the upper lip, nasal cavities, antrum, posterior nares, nasopharynx, velum, root of the tongue, larynx, and trachea. The lesions may be symmetrical, or unilateral with deformity.

Examination—An enormous, hard upper lip with induration in the region of the anterior nares is characteristic, but is totally absent in some patients who have large and typical lesions elsewhere. In the nasal cavities, nasopharynx, or pharynx there may be an indurated thickening or mound—reddish, pink, or whitish in color—more or less obscured by patches of fetid, tenaceous, mucopurulent or crusted secretion. The lesions are obstructive and sometimes are bandlike in form, in the nose or nasopharynx.

In the *laryngeal murmur* the indurations and secretions described in the preceding paragraph may be seen involving the epiglottis and aryepiglottic folds, or ventricular bands, rarely the cords. The glottis is often obscured from view.

Direct laryngoscopy reveals a subglottic and tracheal stenosis with a glazed mucosa, and palpation with the closed laryngeal forceps shows the induration characteristic of the lesion. In the larynx and trachea blood or bloody crusts are not usually seen, and the lesions are not tender to instrumental contact.²

Symptoms—Hoarseness, cough, scanty expectoration of odorous crusts, and foul breath accompanying a slowly developing, painless obstruction to breathing, during a number of years, are characteristic of the laryngeal and tracheal phases of the disease. In a large percentage of the cases there are bulky deformity of the upper lip and fibrotic obstructive masses in the anterior or posterior nares, nasopharynx, or pharynx. Dyspnea may reach the degree of impending asphyxia ill health when it occurs is due to the obstructive lesions and poor living conditions rather than to any toxic factor. Nasal involvement usually adds fetid discharge and a characteristic rhinolalia clausa.

Diagnosis—When accompanying characteristic, bulky, firm, nonulcerated lesions are found in the upper lip, nasal cavities, nasopharynx, larynx, or trachea, the diagnosis is clear, but it should be confirmed by biopsy and fibrotic syphilis should be excluded by a negative reaction to the serologic test. An ample specimen may be taken with no risk of hemorrhage difficult to control. The characteristic

microscopic appearances are described in a previous paragraph. Nests of epithelial cells resembling pearls and dendritic hyperplasia are found, but no other microscopic, nor any clinical, feature of cancer is present to support such a diagnosis. Atrophic rhinitis and tracheitis are similar as to the presence of adherent patches of dried foul secretion, but the thin, shrunken submucosa is distinctive. In both diseases there is a foul odor but that of each is distinguishable by those observers who have experienced both.

Prophylaxis—Though practicing in a locality where the disease has never been recorded the rhinolaryngologist should be aware of the clinical features of the disease so that it can be recognized and reported, whether the patient be foreign born or native. People all over the world are intermingling as never before. All investigators are convinced that the disease is spreading to many countries in which it was previously unknown. Prompt recognition of the disease is therefore important, not only directly as concerns prophylaxis in connection with any particular case discovered, but also indirectly as adding to the sum of knowledge of the disease by which we may learn how to prevent it. At present the only prophylactic means known is residence in a region in which the disease is unknown. It might be added that it is by no means certain that this would protect a native of Poland, for example. Avoidance of touch of the anterior nares with an unsterilized finger has much to recommend it apart from the prophylaxis of scleroma.

Treatment—Tracheotomy usually becomes necessary in laryngeal cases.² Irradiation is the only treatment that has proven remedial, and this only in the early stages of the disease. Minimal doses and utmost caution are necessary if irradiation of the larynx is attempted, because the laryngeal cartilages are extremely sensitive to irradiation. Radium is best for endolaryngeal application. Tracheotomy is advisable before irradiation is begun. The sulfonamides seem to ameliorate the disease by control of secondary invaders.³ Potassium iodide may lessen fetor and crusting but it has no effect on induration. A spray of warm normal salt solution may be used for cleansing purposes in the nose, pharynx, or larynx.

Prognosis and Sequelae—If asphyxia and malnutrition are avoided scleroma is not dangerous to life. Prognosis is not favorable for cure, but the patient can be made comfortable

and able to carry on his occupation. In laryngeal cases the functions of the larynx are impaired permanently in most cases. The course of the disease is long and progress is slow. Spontaneous recoveries after twenty five years have been recorded. Chronic laryngeal stenosis may follow in laryngeal cases.

CHEVALIER JACKSON

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GLANDERS

Glanders is a specific inflammatory disease of the air passages. Synonyms are *malleus*, *equina*, and *farcy*, the latter is applied only to the chronic form of the disease, *farcy buds* are nodular and pustular lesions of the disease occurring under the skin. *Malleus laryngis* is the name of the laryngeal manifestation. Glanders is not a common disease in man. The cause is *Actinobacillus mallei*. The disease is usually acquired from horses or mules, but is communicable from man to man. Routes of infection are through the air, the nose by way of the finger, or through an abrasion of the skin. Whether predisposing causes exist or not has not been determined. Veterinarians, farmers, stablemen, and teamsters are more exposed to infection. The characteristic lesions of glanders are nodules that suppurate and become ulcers. These lesions on mucosal surfaces spread, coalesce, and deepen, in some cases reaching the cartilages with resultant perichondritis and chondral necrosis. The lesions are accompanied by diffuse mucosal inflammation and purulent foul secretions. The mucosa of the nose, nasopharynx, pharynx, larynx, and tracheobronchial tree may be involved in the order given or any of these regions may be involved primarily. The first appearance is in the nose in about 70 per cent of the cases, the larynx and tracheobronchial tree are involved primarily or secondarily in

about 30 per cent of the cases. The *Actinobacillus mallei* is found in the mucosal secretions, pus, and pathologic tissues. The clinical course varies. There is an acute, a latent, and a chronic form of the disease. The latent form may appear a year or more after the exposure, or there may be latent recurrences.

In considering symptoms it is important to remember that the symptomless incubation period may be two days or any interval up to a year or more. The first symptom is the appearance of a nodule anywhere on a mucosal or dermal surface. If the initial lesion is on the skin the mucosal surfaces are involved secondarily after a period that varies within remarkably wide limits. A profuse, blood-streaked, foul, purulent discharge from the nose is accompanied by external redness and swelling, involving nose, cheeks, and lips, suggestive of erysipelas except for lack of a sharp line of demarcation. A pustular rash resembling smallpox may appear on the skin or any part of the body. Nodes may appear in the neck or elsewhere. Cough and foul expectoration are present if the tracheobronchial tree is involved. The acute form is accompanied by chills, fever, malaise, toxic symptoms, prostration, and even coma. The reflected appearances depend upon the stage of the disease. Early, one or more nodules may be found on any part of the larynx except the vocal cords. Later ulcers are seen and they may include the cords by extension. An isolated ulcer may be seen on the epiglottis, only a stump may be left. In later stages the entire lumen of the larynx may be occluded by edema and swollen suppurating ulcerated tissue. This is the stage of perichondritis and necrosis. The diagnosis is not difficult if the disease is suspected. The symptoms and appearances of the lesions strongly suggest syphilis, but the reaction to the serologic tests is usually negative, the *Bacillus mallei* is easy to find in the pus and the tissues. In a guinea pig, inoculation produces typical lesions. The peritoneum should be avoided because peritoneal inoculation may be so quickly fatal that there is no time for a typical lesion to develop. Accompanying lesions in the nose are significant.

Prophylaxis is important. Everyone who comes in contact with horses and mules should be warned of the danger of glanders in nasal discharges and suppurative diseases of these animals. This is imperative because no treatment has been proven efficacious. Chemo-

therapy has not yet had a thorough trial *Prognosis* is difficult. The acute disease has a high mortality rate. Most cases end fatally within a few months. Recoveries occur in perhaps half of the chronic cases, but recurrences may appear after months of disappearance of all lesions. The course of the chronic disease may be as long as ten years.

CHEVALIER JACKSON

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MYCOSES OF THE LARYNX

Mycotic infections of the larynx are usually secondary to mycotic infections of (1) the upper air passages, as in actinomycosis, (2) the pharynx, as in leptothricosis, (3) the mouth, as in muguet, (4) the bronchopulmonary system, as in blastomycosis. Pathologically, most of them are in an ulcerogranulomatous stage when discovered. The diagnosis, prognosis, and treatment are the same as given under "Bronchomycosis." It must be remembered that the, unfortunately, so-called "mycosis fungoides" is not a mycosis. It was given this misleading name because its tumors on the skin sometimes show a contour like a mushroom, which he longs to a different class of fungi. It, also, has been observed in the larynx and has been reported along with other dermatoses that rarely show laryngeal lesions.† (See p. 635.)

CHEVALIER JACKSON

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LEPROSY OF THE LARYNX

Leprosy of the larynx is defined as a chronic specific infective disease with leprosy lesions in the larynx. Involvement of the larynx occurs in about 10 per cent of the cases of the tubercular form of leprosy. It is less common in the anesthetic form. The cause of the disease is

Mycobacterium leprae, an acid fast organism resembling the tubercle bacillus. The route is thought to be nasal and the usual location of the initial lesion is the anterior inferior part of the septum. This is strongly suggestive of digital inoculation. A tropical or subtropical climate is a predisposing cause, but persons infected in tropical countries may develop the disease months after returning to a temperate zone. Primary cases undoubtedly occur in northern climates, though rarely, now, they are still not uncommon in Louisiana and Texas. From the viewpoint of pathology there are three types of the disease: (1) the tubercular, nodular, or hypertrophic, (2) the neural or anesthetic form, and (3) the mixed type, combining both the tubercular and anesthetic forms. It is the tubercular form of leprosy that is usually seen in the larynx, though this form has been found in the larynx of patients with anesthetic lesions in the pharynx and palate. The tissue changes somewhat resemble those of tuberculosis, with nodular infiltration, ulceration, and slow cicatrization. The spread of the disease is by way of the lymphatic channels, which become clogged with the organisms and the products of endothelial proliferation.

Though hoarseness occurs it is one of the late symptoms because the vocal cords are not involved early and, owing to the slow progress of the disease, the cricoarytenoid joints, and the muscles that move them, also are not involved early in the disease. The voice has a muffled quality and may be modified by destruction or cicatricial displacement of the velum, and by deformity of the fauces, lips, and tongue. This deformity may be nodular or cicatricial. Because of sluggish progress, dyspnea develops slowly, and only after years does it become urgent. By that time the patient has learned how to get along with little air. Earlier symptoms are nasal discharge, which is bloody at times. The most conspicuous general symptoms are the nodules, tubercles, and anesthetic areas on the skin, with palpable thickening of the superficial nerves. On examination with the mirror the characteristic lesions to be seen are nodules, granulomas, ulcers, and cicatrices. One or all of these lesions may be present in the progressive stages of the pathologic process. The epiglottis may be ulcerated, destroyed, or cicatrized and distorted. The entire interior of the larynx may be obscured by the infiltration of the arytenoid eminences, the aryepiglottic

folds, and distorted epiglottis. By *direct laryngoscopy* a thorough examination of the ventricle and the subglottic area should be made, in either of these regions typical nodules, ulcers, or scars may be found. The cords may be examined in the cases in which they cannot be seen in the mirror image. They are less often involved than any other part of the larynx, but sometimes they show an ulcer or a cicatrix, usually as an extension from the ventricle along the ventricular floor. The laryngeal structures are not sensitive to instrumental contact. Of primary importance in *diagnosis* is the exclusion of syphilis. The nasal, pharyngeal, and laryngeal lesions have a close resemblance to those of syphilis even to the destruction of the septum, at certain stages there is also a resemblance to lupus and tuberculosis. The diagnostic features of these diseases are given under their respective headings. It should be added that the reaction to the serologic test for syphilis is positive in about half of the victims of leprosy who do not have systemic syphilis. The *Mycobacterium leprae* is abundant in the lesions of leprosy, and the dermal and nerve lesions are unmistakable. Yaws may be excluded by the efficacy of its treatment with salvarsan intravenously administered. (For technic and dosage see under "Syphilis of the Nasal Cavities.")

By way of *prophylaxis* the nostrils should not be touched with unsterilized fingers or thumb by anyone living in places where the disease is known to exist. This is a good rule to follow elsewhere, many serious diseases are thus transmitted. As with all laryngeal diseases the first consideration in *treatment* is the adequacy of the airway. Tracheotomy may be required to prevent asphyxia, but laryngeal obstruction comes on so slowly that the patient learns to get along with little air. No local treatment is known to be of any avail but the local effect of chaulmoogra oil in laryngeal tuberculosis, and the systemic benefit from chaulmoogra oil when used intramuscularly would seem to warrant its use as in laryngeal tuberculosis (*q v*). The general *prognosis* is unfavorable. The recoveries are very few. A fatal termination is not ordinarily reached until after many years and is not then due to the laryngeal condition, unless for the lack of a tracheotomy to prevent asphyxia in the rare cases in which this operation is indicated. The functions of the larynx are all more or less impaired.

CHEVALIER JACKSON

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DEFECTS OF SPEECH

Human speech is essentially imitative. For purposes of simplified illustration it may be compared to phonographic recording and reproduction. The brain records patterns of impressions that are reproduced as speech. The reproduction is more or less modified by a personal equation because the human being is not a robot, but, basically, speech is a reproduction of impressions received through the eyes and the ears, assisted by impressions gained by experience in tactile and muscular senses. The centers, the laryngeal mechanism, and the afferent and efferent pathways involved have been explained in connection with the physiology of the larynx, the phonatory function, the human voice, and the mechanism of speech (Figs. 334, 335).

Defects in speech, collectively called *lalogopathies*, also *mogilalia*, come from imperfections and dysfunction in the various factors by which speech is produced. The most frequently encountered form is stammering or stuttering, called by various names, *alalia syllabaris*, *anarthria literalis*, *balbuties*, and *psellism*. It is characterized by an inability to connect words or syllables together, or consonants with syllables or vowels. No end of controversy has arisen from lack of recognition that there are two great classes of cases, one is a psychoneurosis, the other a structural deficiency. Neither is due to any lack of intelligence. The stammerer of either class thinks clearly and correctly, usually he writes smoothly and without hesitation. A transient form may occur in normal persons when subjected suddenly to fright or emotional shock of any kind, and sometimes this is the start of an established stammering habit, a psychoneurosis. Usually, however, the psychoneurotic form starts from imitation of an example or from perpetuation of an accidental episode of defective speech. The serious and intractable organic form is due to a defect somewhere in or between the central and the peripheral mechanism of speech. Ever

in these cases the attack is precipitated or worsened by attempting to speak before an awe inspiring person or a group of listeners *Logorrhea* (abnormal volubility, or "cluttering") is due to uncontrolled mental activity exceeding the speed of the speech mechanism. In children it is called *paraphrasia precoc*. *Logospasm* is spasmodic utterance. *Embolalia* is the interjection of meaningless words. *Dyslalia*, *alalia*, and *lalopathy* are applied to any defect of the molds of speech, and *laloplegia* means paralysis of these molds. *Rhinolalia* is applied to a nasal tone of voice. It may be due to the passage of too much air (*rhinolalia aperta*) or too little air (*rhinolalia clausa*) from the pharynx to the nose. The morbid anatomy is usually in the velum or the hard palate, or both. More or less of either may be lacking from congenital deficiency as in cleft palate, later in life deficiency may result from disease, such as syphilitic ulceration, either condition may permit passage of too much of the vibrating phonatory air column. The velum may be partially or completely adherent to the posterior wall. The commonest cause of the *clausa* type is adenoids. *Rhinolalia clausa* causes *m* to sound like *b* and *n* to sound like *d*. A *g* sound is given to *c*. *Rhinolalia aperta* gives a dull nasal tone with a sound of air waste. *Diplophonia* and *diphthongia* are applied to doubling sounds as explained in connection with *dysphonia plicae ventricularis*. The falsetto voice is usually voluntary. Occasionally, however, it occurs as a persistent childish voice at and after mutation. *Aphasia*, *aphonia* and *anomia* are elsewhere herein considered.

Prophylaxis of Defects of Speech—Every parent and nurse, and every relative or other person coming in frequent contact with a child, from infancy to adolescence, should understand the basic principle that speech of the child will be an imitation of what he hears. Therefore he should have only the best examples of voice, speech, words, and sounds to follow. This would, in many cases, cure the minor defects of speech, such as lispings (*sigmatism*), and the incorrect use of the *s* or *z* sound (usually the substitution of *th* for *t*). No one should talk "baby talk" to a baby. Children should not be allowed to associate with a stammerer, for example. However, additionally, it is important to realize that it is often not so much that defects are necessarily copied directly from a bad example, but, rather, that the constant impact of a good example would go a long way in

helping the child overcome a minor difficulty. It is deplorable to note, as previously mentioned, that scarcely any parent realizes the extent to which a child can, by example, be enabled to acquire a speaking voice not only free from defect but pleasing in quality, nor do they realize what an asset such a voice could be later in life.

Treatment—Much can be done in the treatment of stammering by the cooperation of intelligent parents. The matters mentioned in the preceding paragraph have a bearing also on treatment. In many cases, especially in children, stammering can be cured by re-establishing the child's self-confidence, and by the mother smilingly and smoothly repeating the difficult words. The stammerer should never be ridiculed, nor embarrassed by direct notice or reproof for the defect. Everything should be done to get his mind away from all thought of the defect of speech. It must always be remembered that there is no lack of intelligence, the mature stammerer knows perfectly well what he wants to say and the words with which to say it, he can write the words fluently, it is only in the mechanism of utterance that he has difficulty. With children it is of utmost importance never to mimic the child's stuttering in his presence. The amusement caused will lead him to think he has said something smart and it will encourage him to repeat it many times until it becomes a habit. Energetic bilateral arm movements, or swaying, coordinated with the words as spoken by the patient will help to bridge the gaps or jump the hurdles in speech. Whenever practicable the child should be placed as early as possible under guidance of a laryngologist who has instructors especially qualified in the overcoming of defects of speech. Some of the problems can be solved only by thorough neurologic or psychiatric study. Other cases such as *linguette* are quite simple as to both diagnosis and treatment. This condition requires only the liberation of the tongue by a minor operation on the frenum. *Rhinolalia aperta* may require a prosthesis or a plastic operation to diminish the opening into the nasal cavities, the *clausa* type may call for the liberation of an adherent velum. It may be said down as a rule to which there are few exceptions that defects of speech requiring operation also need, afterward, carefully planned and continuously applied instruction and education for best results. Slow, careful, interesting teaching—

plans are fundamental. In almost all cases of defective speech there is a necessity to abandon erroneous nerve cell habits and to acquire new and correct habits. Daily breathing exercises are essential. The various forms of abnormal speech sometimes associated with a psychosis are a relatively minor part of the treatment of the patient by the attending psychiatrist.

Prognosis—Stammering is curable in nearly all cases if the treatment is undertaken in childhood. Later in life it is more difficult, but even in adults most cases can be cured. Slight and temporary relapses may occur under stress. Rhinolalia and tongue-tie can be cured by prosthesis or operation. Tongue-tie and lipping are curable. In general it may be said that defects present in childhood are curable if treated in childhood. The prognosis of rhinolalia coming on late in life depends upon the cause.

CHEVALIER JACKSON

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PSYCHOSOMATIC APHONIA

Psychosomatic aphonia may be defined as laryngeal manifestations resulting from psychic trauma. *Psychosomatic aphonia* is applicable when aphonia includes absence of whispered voice. The condition is inaccurately referred to as *voluntary aphonia*, *hysterical aphonia*, *shell shock*, *neurotic aphonia*. The term *functional aphonia* is open to the objection that there is always a question as to the justification of the subdivision of maladies into organic and functional classes.

Psychosomatic aphonia is relatively common in times of peace. A large number of cases develop in wartime because of the prevalence of the predisposing and exciting factors to which civilians as well as military personnel are exposed, such as those of World War II.

Etiology—The most common exciting cause is an emotional shock, a psychic trauma, with direction of attention to the voice or larynx. For example, a person may be struck dumb or knocked speechless or be speech bereft

or speech bound by an explosion. In a person of average stability the voice is quickly recovered and indefinitely retained. In a person of psychopathic instability a morbid state with mental fixation on the loss of voice may be initiated. The fundamentals of this simple and common example underlie most of the cases, but the great number of obscuring and confusing incidentals may render clear analysis of the minor etiologic factors difficult. Instability may be congenital or acquired or may be produced in a previously normal person by such factors as fatigue, exhaustion, insufficient rest, anxiety, fear, nostalgia, avitaminosis, dietary deficiency, malnutrition, incessant noise and toxic conditions. The factors fixing the physical manifestations on the voice or larynx may be equally difficult to analyze. They may be from subjective experiences such as a laryngitis or objective suggestion as in meeting a person with laryngeal or vocal trouble. The just mentioned analytic difficulties apply to many cases in civil life under conditions of peace. In the relatively large number of cases of *psychic laryngeal trauma in warfare* affecting civilians and military personnel the exciting and predisposing factors are obvious.

Pathology—Though it is usually difficult or impossible to demonstrate organic tissue change in these cases it must be conceded that the former conception of purely functional disturbance without any organic basis cannot be logically maintained in the light of clinical experience. The initial subjective suggestion in many cases in our clinical experience was an ephemeral adductor laryngeal paralysis (*q.v.*) due to vascular cortical disturbances, chiefly anemia from emotional shock, fright, swooning, concussion or the like. It seems probable that many cases of psychic laryngeal trauma are thus initiated. In many other cases memory of some such episode may suggest the larynx for the somatic localization when psychoneurotic conditions develop after an intervening period, even years of normal life. The fundamental pathologic factor that must be recognized for efficient treatment is the existence of one of the many forms of a psychoneurotic state. Usually underlying the laryngeal somatic manifestation there are a conflict, a maladjustment to reality and an escape mechanism. It is an injustice to the patient and especially so in case of a veteran to call this psychoneurosis hysteria.

Symptoms.—The patient is obsessed with the idea that he has lost his voice. When asked a question he may simply shake his head or may make prodigious movements of the facial muscles without uttering a laryngeal sound. Usually he makes no attempt to whisper, although in some rare cases the disability takes the form of inability to speak louder than a whisper. The general symptoms of psychoneurosis may be latent, mild, or severe. In the cases following explosions, wrecks, prolonged bombing, bombardment, and battle conditions there may be profound systemic disturbances, depression, listlessness, spasms, tremors.

Reflected Appearances.—On quiet breathing, or deep inspiration, the laryngeal appearances are normal. When, however, the patient is requested to say "Ah" the cords do not approximate, they may flutter slightly, or lie in a mid-way position. Air may or may not come up through the larynx. The patient may simply shake his head. The essential thing is to make him phonate. This is accomplished in most cases by asking him to cough, while the mirror remains in place. The cords are seen to approximate, thus making the cough phonatory. In the few cases in which the patient does not cough when requested, he can be forced to cough by placing a few drops of sterile water into the larynx with a laryngeal syringe under guidance of the eye and the mirror. Of course local anesthesia must not have been used. In some cases, especially in victims of explosions, or in veterans who have gone through terrific ordeals, other images may be noted, as hereinafter mentioned.

Direct Examination.—This is not required except in cases in which no approximative movement of the cords occurs during many repetitions of the just mentioned examination with the mirror. In such rarely encountered cases there will be plenty of reflex movements excited by direct laryngoscopic examination without any anesthesia, general or local.

Diagnosis.—The existence of laryngeal somatic manifestations of a psychoneurosis characterized by apparent aphonia and apathyria, is easily and definitely made by the examinations described in the two preceding paragraphs. The diagnosis as to the causative psychic trauma may all come out clearly in the course of an ordinary history as, for example, when a patient attributes the loss of voice to a terrifying explosion, or when a veteran tells of his ordeals,

but in other cases to find the cause of the conflict and maladjustment may require all the ingenuity and patient conversational search of the neurologist's usual methods of eliciting a case history. Previous injuries, minor laryngeal ailments of self, companions, or family, and similar features may be relevant. Of course, in all cases, complete systemic examination is necessary to find or exclude serious mental derangement and organic disease, syphilis and tuberculosis especially. In pulmonary tuberculosis there is often aphonia, but not apathyria. The voice is weak and may disappear but the molds of speech produce words from the fricative sound of air passing between the inactive cords, or there may be obvious and partly successful effort at approximation. Muscular disability is often present in the early stages of pulmonary tuberculosis without any tuberculous lesion in the larynx. One condition always requires exclusion when there are present the previously mentioned appearances in the mirror denoting failure to approximate and phonate except on voluntary or induced cough, namely, *malingerer*. This is a totally different thing. The malingeringer has no psychoneurosis. He is wilfully lying in claiming he cannot talk, and he is doing it to escape duty, or to perpetrate a fraud such as a claim for workmen's compensation or for insurance indemnity. He knows perfectly well he can phonate. This is really a *voluntary aphonia* and this term is preferable to the word "malingerer." Differential diagnosis depends largely on three things that indicate malingering, namely, the absence of a psychoneurosis, the finding of the objective, and a stubborn lack of cooperation in treatment.

Varieties of the Disease and Association with Other Diseases.—The laryngeal manifestations are often associated with other somatic evidence of a psychoneurosis, or with somatic injury, or with other illness. In the case of veterans who have undergone long ordeals of continuous bombing of bombardment, or of battle, there may be an attempted phonation—(1) the elliptic glottic silhouette of thyro arytenoid disability, (2) approximation of the anterior part of the cords, leaving the triangular glottic silhouette posteriorly that denotes disability of the arytaenoides, (3) spastic contraction of all the closing muscles of the larynx. All of these conditions simulate motor paralyses (*q*). The difference is that in the case of psychic trauma complete recovery occurs under proper treat-

ment *Myasthenia laryngis* may coexist with psychic trauma

Treatment.—It is of utmost importance to avoid telling the patient, and to prevent anyone else telling him, that he is wilfully shamming. His ailment must be taken seriously, but not as a serious illness. He must be made to realize that his ailment is not dangerous and is quite curable. His confidence and cooperation in readjustment to reality must be obtained. He is lacking in determination to get control of himself and exert the energy necessary for this self-mastery. This deficiency in morale, as it might be called, must be made up by creation of inspiration and the will to get well and to phonate. All this part of the treatment is in constant use by neurologists in the treatment of various forms of psychoneurosis. Additionally there must be brought to the treatment of the case deep-breathing exercises practiced regularly. Then the patient is told to take a deep breath and hold it, a moment later he is told suddenly to release it with a cough. Then to release with a groaning sound. This is pointed out to the patient as a beginning of a voice. In some cases traction on the tongue will enable the patient to get a start in phonation by the sagittal drag on the epiglottis and laryngeal tissues. Some patients get a start by giving a thump to the sides of their thorax with both elbows synchronously with a grunt, which seems to add force to cortical impulses. Various other methods may be used, but all treatment must be carried out with inspiring confidence and cheerfulness. In all cases, medical care and management are essential. Avitaminosis and other dietary deficiencies as well as all environmental factors in impaired health must be eliminated. The long-continued administration of a daily excess of a good mixture of vitamins, as supplied by one of the reliable manufacturers of pharmaceuticals who maintain biological laboratories, is helpful, especially so when used synchronously with a balanced diet.

Prognosis.—In practically all cases a cure can be obtained by proper treatment. Recurrences are not infrequent but they are equally amenable to proper treatment.

CHEVALIER JACKSON

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HYPERESTHESIA AND PARESTHESIA OF THE LARYNX

An abnormal sensitivity or sensation referred to or referable to the larynx is known as "hyperesthesia and paresthesia of the larynx." It is recognized that wide variation exists within normal limits of general sensitivity and stolidity.

Abnormal sensations referred to the larynx are common. Most of the cases arise from one or more of the numerous causes of psychoneurotic conditions, such as morbid introspection, hysteria, and hypochondriasis. These conditions often have an organic basis in systemic diseases altogether unrelated to the larynx. Psychic trauma (*q v*) is the most common cause.

There is no laryngeal structural tissue change, though some preceding laryngeal ailment or trauma may have fixed the psychoneurotic attention to the larynx. In a few cases there may be a relatively insignificant minor laryngeal ailment that fixes the attention of the patient on the larynx but does not cause the ailment of which he complains. The patient may complain of such sensations as tickling, jaggling, burning, itching, tightness, pain, soreness, or rawness. Perhaps the most frequent complaint is of a constant and compelling desire to cough or to clear the throat of secretions or of a foreign body. The complaint may be absurd as of worms crawling or insects stinging. *Diagnosis* depends upon exclusion of abnormality in the larynx. Psychoneurosis is a condition readily determined by neurologic methods of study as given in works on that subject. Malinger is to be excluded. It is not a psychoneurosis, but a deliberate attempt to escape duty or perpetrate a fraud. The objective will be found.

In the *treatment*, first and most important, it must be realized that a psychoneurosis is a disease to be handled by appropriate therapy. This is not in the province of the laryngologist. The patient should never be accused of shamming. His ailment must be taken seriously. Local treatment of any minor ailment of the larynx usually serves only to aggravate the disease by promoting fixation of attention on the larynx. (Further suggestions for treatment will be found under "Psychopathic Aphonia.") In practically all cases the patient can be cured. Recurrences are not infrequent but these are amenable to treatment.

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ICTUS LARYNGIS

Ictus laryngis is a sudden attack associates with or simulating glottic spasm *Laryngeal syncope*, *laryngeal vertigo*, and *laryngeal epilepsy* have been used synonymously *Laryngeal cataplexy* would seem more appropriate for some cases.

Ictus laryngis is not a common ailment The cause is unknown, and no morbid anatomy has ever been definitely associated with the ailment The theories do not have the support of clinical facts in any large group of cases of the disease Remarkably little has been added to our knowledge of the ailment since it was discovered as a clinical entity by the great clinician, Charcot, in 1876 Almost all the cases occur in men between thirty and seventy years of age The momentary unconsciousness, the hitting of the tongue, and the slight convulsive movements present in some cases suggest a relation to epilepsy When these are absent the attack is rather suggestive of cataplexy No alternate attacks occur, however, without the laryngeal phase

The ictic attack usually commences with a paroxysm of cough which terminates suddenly with a clicking sound like a hiccough, the patient has a subjective sense of dizziness, he loses consciousness and falls Within less than a minute consciousness returns, though mental confusion may continue for a few minutes There may be either cyanosis or pallor In some cases there are hitting of the tongue and convulsive muscular movements in the face and extremities In some instances there is no unconsciousness, only confusion or bewilderment, which the patient describes as giddiness or dizziness because he does not know of a more accurate word

There seems to be no record of a laryngeal examination during an attack, but examinations after an attack in our cases revealed nothing abnormal We have frequently examined the larynx during hiccough, which has a similar sound The cords snap together and the ventricular floors sink

The symptoms described are diagnostic, but thorough general medical and neurologic examinations should be made in every case and these examinations should be repeated at intervals of a few months Some of the organic cerebrospinal diseases present a similar syndrome, especially tabes (*qv*) In this disease it is seen as a laryngeal crisis, which may or may not be followed by a bilateral laryngeal paralysis

There is no known treatment that will prevent an attack The patient should be thoroughly studied and any indications for betterment of his general health, or for elimination of any unfavorable condition of environment that may be discovered, should receive appropriate attention General or local treatment for the attack is unnecessary If there are abnormalities in the larynx they should, of course, be treated according to indications Asphyxia has been reported in patients who had previous attacks of ictus laryngis but there may have been other factors in such cases There are no contraindications to tracheotomy when it is really indicated Recovery is the rule Recurrences may be as frequent as daily or as infrequent as once in a lifetime There is no regular periodicity

CHEVALIER JACKSON

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SPASM AND OTHER MOVEMENTS OF THE LARYNX IN ADULTS

Spasmodic closure of the larynx may involve only the glottis, but usually it involves all of the laryngeal closing musculature as described in connection with physiology of the larynx In any case both degrees of spasm are usually present at times The commonest form of spasm is a manifestation of the phylogenetically earliest function of the larynx, the reflex protective spasmodic closure and tussive expulsion of a foreign body It may be a few drops of water or of pharyngeal secretion, but, not being air, it is foreign to the lungs and must not be allowed to enter This is normal

Laryngeal tic, or, as it is sometimes called, *diaphragmatic tic*, is a snapping together of the vocal cords causing a sound called a "cry" though it is painless. It is similar in mechanism to the *clicking in singultus* and is passive so far as the larynx is concerned, the sudden, more or less phonatory sound is due to the forcing together of the cords by the sudden inrush of air which makes pressure on the floor of the ventricles (Fig. 357). A *spasmodic laryngeal cough* is the reverse of this mechanism, being active not passive adduction. It is seen in the psychogenic neurosis called "hysteria," in psychic trauma (*q v*), the barking cough of puberty, tetanus, and hydrophobia. A traumatic condition that has been erroneously called a neurosis is the *megaphonia*, or *aphonia*, mostly affecting public speakers, ministers, lawyers, evangelists, stump speakers, and others who abuse the larynx by prolonged exhortation. The voice may be unaffected during conversation but on attempting public speaking there is a spasm of the adductors and tensors, that usually continues as long as the effort to speak is continued. A parallel condition is seen in professional singers, they have little or no trouble in subdued conversation, but any attempt to sing precipitates a phonic spasm. Both these conditions (dysphonia spastica) are *occupational disabilities of the larynx*, both are due to indirect trauma, they are of the same prognosis and they require the same treatment as myasthenia laryngis (*q v*). Our clinical records show cases of bilateral laryngeal jerking movements in tetanus, chorea, and hydrophobia. Any of the conditions subsequently enumerated herein as producing laryngeal paralysis and syndromes may be associated with spasm, usually prior to the paralysis. These may be central lesions, or lesions of the ninth, eleventh, and twelfth cranial nerves near the jugular foramen, but especially of the tenth, the vagus, which contains both the efferent and afferent laryngeal neurons (Figs. 334, 335). In *tetanus* there are not only the laryngeal motor and sensory paralyses elsewhere herein mentioned but typical spasm in the form of *laryngeal crises* early in the disease and arrhythmic jerking and incoordination of cordal movement. Our clinical records show tremors of the vocal cords in multiple sclerosis, syringomyelia, paresis, encephalitis, parkinsonism, alcoholism, and hyperthyroidism. Laryngeal tremors are usually bilateral and they are symmetrical except when incoordination is

present. Fine tremors of the cords are easily overlooked, coarse tremors must be distinguished from the normal alternate hesitation and movement that is present in many individuals.¹ Jerking movements of the cords or arytenoids, such as occur in chorea, are easily recognized, so are the to-and-fro movements of the cords that occur in cases of pharyngeal nystagmus.

CHEVALIER JACKSON

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PARALYSIS OF THE LARYNX

The term "paralysis of the larynx" denotes laryngeal loss of motion or sensation. *Laryngeal palsy* is a synonym. *Recurrent laryngeal paralysis* is often used synonymously, but it can be correctly applied only to certain cases. *Hysteric paralysis* is a misnomer.

Paralysis of the larynx may be sensory or motor. The latter form has various names according to muscles affected. The disease is often spoken of by the location of the lesion, as bulbar, central, peripheral, and also by names of various syndromes. Motor paralysis may be spastic or flaccid. The various forms encountered clinically require separate consideration. The usual confusion in these complex subjects will be avoided if the simplified schemata shown in Figures 334, 335, and 398 be viewed in connection with the physiology of the larynx, the innervation of the speech mechanism, and the table of muscles and nerves given under "Paralyses in Relation to the Esophagus."

Sensory Paralysis of the Larynx.—This may be defined as loss of sensation and reflex action due to lack of sensory innervation of the laryngeal mucosa. *Anesthesia of the larynx* is a synonym not always applicable.

Sensory paralysis of the larynx is often present but is usually overlooked. The most frequent cause is impairment of the functional continuity of the trunk or of the internal branch of the superior laryngeal nerve. Above the giving off of this nerve from the vagus the causes are the same as those hereinafter enumerated.

for the adductor and abductor muscles. Anesthesia of the larynx occurs in association with various bulbar lesions. It is frequently present in tabes. Any lesion involving the lower neurons above the level of the giving off of the superior laryngeal nerve will produce a paralysis of the sensory or motor function of this nerve, usually both. These lesions are discussed along with those involving the motor neurons of this nerve and the inferior laryngeal nerves. Apart from toxic neuritis, sensory function of the larynx may be temporarily paralyzed by local application of drugs, especially cocaine. This does not affect the motor branch of the superior nerve, except as to abeyance of the excitatory afferent impulses that normally activate reflex motor functions in the muscle (cricothyroid) to which the motor branch of this nerve is distributed. The motor function is an important part of the cough reflex arc and all the other laryngeal muscles, innervated by the inferior laryngeal nerves, participate in the abeyance. A similar but more dangerous abeyance is produced by opium derivatives. We have seen a large number of such cases. Metals may cause sensory laryngeal paralysis by producing a toxic neuritis. We have seen one such case due to lead. The selective action of systemically absorbed metallic poisons for particular nerves has never been satisfactorily explained. The same may be said of diphtheritic toxins, they were the cause in two of our cases. In another case systemic syphilis was the cause. In three other cases, complete laryngeal anesthesia occurred in the fourth week of typhoid fever. In bulbar lesions of tabes, myasthenia gravis, syringomyelia, and multiple sclerosis, as well as in cases of hemorrhage, thrombosis, tumors, and other lesions in the region of the jugular foramen (Figs 335, 398), anesthesia of the larynx is often present but in these conditions it is usually overlooked for want of a local laryngeal test.

Anesthesia of the larynx may be symptomless unless a test is made. If the injury to, or disease of, the sensory lower neurons of the superior laryngeal nerve involves the nerve of one side only, the nerve of the other side will preserve sensation because of *bilateral distribution*. When both sides are paralyzed the absence of sensation might not be noted by the patient. There would be, however, loss of the protective reflex function of the larynx, permitting foreign matter, especially pharyngeal secretions, to

enter. Cough would occur after other afferent pathways, those of the tracheal or bronchial mucosa, were reached. Paroxysms of cough usually occur on swallowing food or liquids.

The only way to make the *diagnosis* of sensory laryngeal paralysis is by test. The simplest test is to pass the direct laryngoscope without anesthesia. The absence of the laryngeal mucosal reflex is obvious to anyone accustomed to doing direct laryngoscopy without any anesthesia, general or local. The mirror may be used to guide the touch of the laryngeal probe but, if there is no pathologic anesthesia of the tongue or fauces or pharynx, a touch of the probe on any of these parts may cause obscuring reflexes such as gagging. Elimination of the sensitivity of these parts by cocaine or other drugs is difficult because of the proximity of both superior laryngeal nerves to the mucosa of the laryngopharynx. In making the diagnosis of sensory paralysis of the larynx the presence of other paralyses of the larynx, of the pharynx, palate, tongue, and lips should not be overlooked (see under 'Syndromes Associated with Laryngeal Paralysis').

The *treatment* of sensory paralysis will be clearly indicated when the cause of the paralysis is found in the particular case.

Motor Paralysis of the Larynx—The term "motor paralysis of the larynx" denotes loss of power of laryngeal movement due to impaired innervation. Laryngeal motor paralysis may be unilateral or bilateral, *spastic* or *flaccid*. Motor paralysis may be classed as *adductor*, *abductor*, or *tensor*, according to the muscle groups affected. When all groups are paralyzed and the tonus is gone it is called *complete paralysis*. The term "cadaveric" has been so loosely applied to different conditions that it should be dropped to prevent confusion.

Clinically as well as for clearness of presentation the laryngeal muscles are best considered in two vital groups, namely, (1) the closers of the larynx, which protect the airway from the intrusion of foreign bodies, especially food on swallowing; (2) the powerful openers of the larynx, which maintain a vital airway. These two vital groups are directly opposed to each other in a wonderfully balanced, cooperative, mutual resistance that automatically maintains an airway, regulates intrathoracic pressure, circulatory pressure, and the acid base balance, and other important functions. Additionally it is at all times called upon to respond cooper-

atively in phonation and in the cough reflex (see "Physiology of the Larynx") This balance between the opening and the closing muscular groups is fundamental to an understanding of certain forms of paralysis, it depends upon normal activity on the part of both groups of muscles When innervation of one group is impaired, the other group unopposed becomes overpowering In considering closure we must remember that closure of the larynx and closure of the glottis are not the same thing During swallowing both close synchronously, but in phonation the glottis closes while the larynx remains forcibly held open In closure of the larynx (not solely of the glottis) muscles of the pharynx, epiglottis, and tongue participate as described in connection with the swallowing function (*q v*) We are concerned here with the laryngeal muscles The closing muscles, called *adductors*, are (1) the crico arytaenoides laterals, which brings the cords parallel by a forward pull, (2) the interarytenoid, which closes the posterior part of the glottis by approximating the arytenoid cartilages, (3) the thyro arytaenoides internus, which draws together the vocal processes, cooperating with the arytaenoides it closes the cartilaginous glottis, (4) bands of muscular fibers in the aryepiglottic folds, which aid in closing the *supracordal larynx* All the closing or adductor muscles are innervated by the adductor neurons in the inferior laryngeal nerves These two groups, by synchronously and strongly opposing each other, assist in phonation especially in the control of pitch In this phonatory counterpull they are assisted by a third muscular element, the pair, right and left, of cricothyroid muscles often called the external "tensors" To get a proper conception of the effect of paralysis of one or both of the cricothyroid muscles, we must have a proper conception of the action of this muscle, and of the mutual relations of the cricoid and thyroid cartilages These cartilages rock on a pair of pivotal joints Sometimes one cartilage is muscularly fixed for the other to rock upon, at other times they exchange duties This alternate rocking can be felt and should be part of the training of the fingers of every laryngologist During swallowing (1) the thyroid cartilage is fixed (by the muscles attached to its outer surface and upper edge) while the cricoid rocks For phonation (2) the cricoid is fixed by the backward pull of the cricopharyngeus, while the thyroid cartilage rocks forward under

the pull of the cricothyroid muscle This rocking increases the distance from the anterior commissure to the posterior commissure, in other words, the glottis is elongated This elongation and the accompanying fixation enables the thyro-arytaenoides to harden the edges of the cords to raise pitch in phonation by diminishing cordal elasticity The elongation is not enough to raise pitch, it amounts only to a taking up of the slack It is not comparable to the tensile raising of pitch of a guitar string (see p 423)

The *abductor* group of muscles is composed of the crico arytaenoides postici, one on each side, and the cricopharyngei, also one on each side They slide the arytenoid cartilages outward and at the same time tilt the vocal processes of these cartilages backward, the combined effect of these movements is to open the glottis by this abduction of the cords Normally they hold it open against the counter pull of the adductors by reason of greater power They relax coordinately when the closing muscles approximate the cords for phonation The abductor (opening) muscles are innervated by the abductor chain of neurons in the inferior laryngeal nerves

In addition to the adductor and abductor groups of muscles there is a third group, which is concerned with the regulation of the elasticity or hardness of the vocal cords for control of pitch, the pair of cricothyroides, known as the external tensors, and the pair of thyro arytaenoides, often called the internal tensors (When the word "tensor" is used in this book it is with the semantic limitations mentioned above and under "Physiology of the Larynx")

CHEVALIER JACKSON

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- 1 Jackson Chevalier, and Jackson Chevalier L. Diseases and Injuries of the Larynx, ed 2, New York, The Macmillan Company, 1942

PARALYSIS OF THE CRICOTHYROID MUSCLE

Synonymous with the term "paralysis of the cricothyroid muscle" are *paralysis involving the superior laryngeal nerve*, *tensor paralysis*, and *paralysis of the external tensors*

Compared to the inferior laryngeal nerves, the superior pair are much less frequently affected. Probably this is attributable to the direct course of these nerves, they are given off the vagus at the level of the larynx whereas the neurons for the inferior pair continue in the vagi all the way down the neck, and up again, as inferior laryngeal nerves, through an area subject to many kinds of morbid processes (Fig 334). As an isolated condition cricothyroid paralysis is rare, as part of a complete paralysis (*qv*) it is relatively frequent.

Etiology and Pathology.—Any lesion affecting the neurons of the motor branch, or of the trunk, of the superior laryngeal nerve will cause paralysis of the cricothyroid muscle. The lesion may be operative, traumatic, diphtheritic, toxic,

deus, which, being innervated by the inferior laryngeal nerve, is not involved in the paralysis. If the causative lesion involves the lower neurons, as it usually does, the wrinkling is greatly increased because the paralysis is of the flaccid type. One or both nerves may be involved, if both, the tonus may be gone. When only one nerve is affected the cords are on a different level and askew.

Reflected Appearances.—When the pair of cricothyroids alone is affected the appearances reflected in the mirror are as shown in Figure 377. Abduction and adduction are normal. When only one of this pair is involved the long axis of the glottis is askew (Fig 394). The cords in cricothyroid paralysis are seen to be only slightly if at all relaxed if the lesion is above the

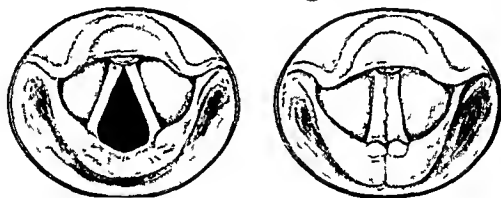


Fig 377—Reflected appearance in a case of motor paralysis of the superior laryngeal nerve. Both cricothyroids being paralyzed the thyroid cartilage cannot be rocked forward on the cricoid to give phonatory fixity against the counter pull of the thyro-arytenoid. The inspiratory abduction as shown at the left, the good adduction as shown at the right, and the wrinkling by the thyro-arytenoid—all these show unimpaired activity of the muscles supplied by the inferior laryngeal nerve.

neuritic, adenopathic, or neoplastic. Cephalad from the vagal giving off of this nerve the etiology and pathology are the same as given for this region in connection with the subject of the involvement of the inferior laryngeal nerves (*qv*). The motor paralysis may be spastic or flaccid. The paralysis of the cricothyroid muscle prevents the function of this muscle in fixing the thyroid on the cricoid cartilage to resist the pull of the thyro-arytenoid muscle. Lacking this resistance this muscle cannot barden the edge of the cord to control pitch, and in the effort to do so the edge of the cord is wrinkled. The statement in the textbooks that the cord is wavy as the result of "failure to be put on the stretch" is inaccurate. The cord is wrinkled by the action of the thyro-arytenoid

nuclei ambiguus (always bilateral), quite flaccid if below these nuclei. Infra-nuclear paralysis may be either unilateral or bilateral, but, usually, is unilateral.

Appearances on Direct Laryngoscopy.—In the laryngoscope the cords are seen to be wavy. When the tip of the distal end of the laryngoscope reaches the anterior commissure the waviness disappears. The passive mobility test shows free movement at the crico-arytenoid joints. In unilateral cricothyroid paralysis the waviness is one-sided and the long axis of the glottis is askew. The cords are seen not to be on the same level.

Symptoms.—The voice is weak, rough, and of lower pitch than normal for the particular patient. The respiration is wheezy as heard at

the open mouth during mirror examination. These symptoms are marked when the cricothyroid paralysis is bilateral, and there is then added the tracheobronchial cough from foreign matter, such as food, fluids and pharyngeal secretions, which may pass through the anesthetic larynx that lacks its normal protective reflex action.

Diagnosis.—The mirror appearances described in a preceding paragraph are diagnostic. In case of a bilateral lesion above the origin of the internal (sensory) branch of both superior laryngeal nerves (Fig. 334), anesthesia of the mucosa would be easily detected by direct laryngoscopy without anesthesia. Prodding with the closed laryngeal forceps is seen to produce no reflex cough. A unilateral lesion would not produce anesthesia because of the overlapping of the opposite sensory nerve distribution. Ankylosis of the cricothyroid joint can be detected by palpation. Diagnosis as to location of the lesion is the same as that herein given for sensory and motor laryngeal paralysis. Tests for syphilis (*q v*) are always of primary importance. When the reaction to the blood test is negative the spinal fluid test should not be omitted. If the reaction to either test is positive the lesion may be central or peripheral (Fig. 334).

Treatment.—Treatment as to kind and advisability will depend entirely upon the cause of the paralysis. If the causative lesion be cured the laryngeal paralysis may not be helped thereby, but the lesion may call urgently for treatment for other reasons. Treatment of the laryngeal muscular disability is the same as given for adductor and abductor paralyses.

Prognosis.—The paralysis of one cricothyroid muscle or both of these muscles in itself involves no danger to life. Causative lesions may or may not be serious. Recovery of function is rare no matter what the cause, but there is, in many cases, improvement from compensatory adjustment by the laryngeal musculature.

CHEVALIER JACKSON

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ADDUCTOR PARALYSIS OF THE LARYNX

Paralysis of the glottic closing muscles due to impairment of their innervation is known as adductor paralysis of the larynx. Literary confusion has resulted from misapplication of the name to cases of crico arytenoid arthritis, psychic trauma, malingering, and other unrelated conditions.

Adductor paralysis, properly so-called and as a sole condition, is rare, but we have seen a number of such cases. If the larynx were always examined in cases of concussion of the brain probably many cases of adductor laryngeal paralysis would be noted. As a part of a complete paralysis it is relatively common.

Etiology.—Adductor paralysis may be caused by any central lesion located as described in the subsequent paragraph. In five of our cases the cause was cerebral concussion from blows on the calvarium, in two other cases the cause was syphilis and cerebral complication of typhoid fever, respectively.

Pathology.—Adduction usually is a voluntary movement requiring cortical impulses. A supranuclear (above the nuclei ambigu) lesion must involve the fibers (upper neurons) of both sides, to produce a paralysis because partial decussation of these fibers enables one side to act for both (Fig. 334). To involve the widely separated cortical executive centers a pair of exactly symmetric lesions, or one huge lesion, is necessary. A lesion at or near the decussation would not necessarily be large. Any lesion above the nuclei ambigu involves upper neurons, hence the resulting paralysis is of the *spastic* type, because the upper neurons carry inhibitive as well as voluntary motor impulses. Hence, not only is executive movement lost, but after a variable period there comes on a hyper-tonic rigidity of the affected muscles. Upper neuron lesions affect groups of muscles, because, in these neurons, movements (not particular muscles) are represented. In the traumatic cases that we have seen there was concussion of the brain due to one or more blows on the calvarium which presumably had jostled the underlying cells in the motor cortex in this area. In one of these cases of adductor paralysis, when seen again at the end of two months, both cords were in the position of complete paralysis with a history of a period of severe dyspnea and improved voice followed by better-

ment of breathing and husky voice. This was an exception to Semon's rule (*q.v.*) When adductor paralysis is associated with a posticus paralysis and loss of tonus (Fig. 396) it is probable that widespread degeneration has involved all the motor and sensory neurons that normally innervate the larynx. This widespread loss of innervation may come on at once as in the just mentioned case or be a slowly spread

affecting the adductor neurons on one side only, leaving the abductor neurons of that side unaffected. One such case under our observation occurred in a syphilitic man aged sixty-four years. It seemed probable inferentially that the adductor neurons to the other adductor muscle, the arytaenoideus, were also affected but this could not be determined because the observed perfect movement of this muscle could have

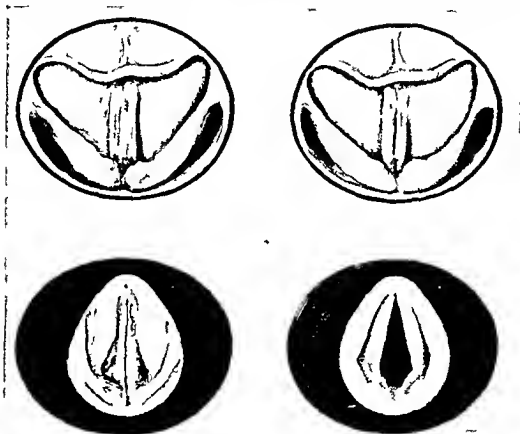


Fig. 378.—At the left above is the reflected appearance on inspiration in a case of median paralysis the canula being corked. The cords are forced tightly together instead of being widely separated as they would be normally. At the right above is the position on expiration the cords are slightly blown apart by the expiratory current. The voice was good but both posticus were paralyzed so the patient could not open his glottis for breathing. At the left below is the appearance on direct laryngoscopy. At the right below is shown the position to which each arytenoid in turn could be displaced in the passive mobility test. The closed forceps with which the passive mobility was demonstrated are omitted so as to give an unobstructed view of the range of passive mobility.

ing degeneration. We have observed both types. This state of total loss of innervation is properly called complete paralysis (*q.v.*) It is rare as a unilateral condition but we have seen a few cases in which this appearance was due to bilateral paralysis of the arytaenoideus. Unilateral adductor paralysis is never complete because the arytaenoideus cannot be unilaterally paralyzed (Fig. 379). In such cases it is justifiable to assume there has been a lesion

been produced by the adductor neurons in the unpaired inferior laryngeal nerve of the other side. There is overlapping of distribution.

Reflected Appearances.—The fundamental characteristic of adductor paralysis is that the cords are paralyzed open (Fig. 379). That is to say they are in an abducted position but cannot approximate so as to cut the air column into puffs and thus cause a sound (see under

"Mechanism of Normal Speech," p 423) A casual inspection with the mirror might mislead an inexperienced examiner into believing nothing abnormal is present, but careful study will reveal the fact that the cords are not so widely separated as in full abduction and they have a firm appearance suggestive of spasticity or hypertonus. In one of our cases of spastic paralysis there was an almost imperceptible momentary twitching of the edge of the cords. On asking the patient to say 'E e e-e' the cords fail to approximate, they may or may not flutter slightly, the arytenoids may show slight movement, but their normal sliding movement of adduction is obviously lacking, a blast of air comes through the open glottis, but it makes only a fricative sound. In the cases in which the

Appearances on Direct Laryngoscopy.—On the approach of the distal end of the anterior commissure laryngoscope passed without anesthesia, general or local, the appearance is quite striking. Instead of the sudden intermittent snapping shut of the glottis on cough it remains constantly open, in the inspiratory position, and the breath comes through on coughing, with little sound. In case of adductor paralysis involving the arytaenoides only, there is a triangular chink posteriorly and there is a whistling sound when air is forced through it (Fig 379).

Symptoms.—The characteristic laryngeal symptom is aphonic voice with air waste. If the patient forces the expiratory blast his molds of speech will afford him a good loud stage

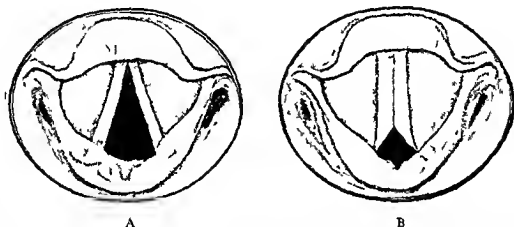


Fig 379 —Sketch illustrating two types of bilateral adductor paralysis. At the left the appearance is that of inspiration but on attempted phonation there was no approximation. At the right the triangular glottic silhouette posteriorly is the typical appearance in paralysis of the interarytenoid muscle which normally closes this cartilaginous part of the glottis. It is always bilateral. It forms part of the image in complete paralysis (Fig 396).

adductor paralysis is accompanied by paralysis of the abductors and "tensors" the tonus is gone and the typical image of the complete position is seen (Fig 396). We have seen adductor paralysis, with the arytaenoides uninvolved, occur as a postoperative condition of short duration as mentioned under "Bilateral Incomplete Paralysis of the Larynx." For paralysis of adduction of the whole length of the glottis the arytenoid muscle must be paralyzed also. When this muscle is paralyzed and the laterales are unaffected there is a triangular chink at the cartilaginous glottis (Fig 379).¹ It is always bilateral because the arytaenoides, having bilateral innervation, would be innervated from the unaffected inferior laryngeal nerve of the other side.

whisper by molding the fricative sound of air passing through the glottis. Cough is of the wheezy inefficient type because of lack of glottic cooperation in raising pressure to produce the explosive release (see under "Physiology of the Larynx"). There may be accompanying symptoms in regions other than the larynx. Some of these are paralytic and are explained under "Syndromes Associated with Laryngeal Paralysis."

Diagnosis.—The diagnosis is easily made by mirror examination, as illustrated in Figure 379. It is essential to exclude the possibility of functional aphonia due to psychic trauma (q 1). This is readily and conclusively done by three simple mirror tests, namely, (1) ask the patient to say "E-e-e-e", (2) ask him to cough,

(3) make him laugh—each of these while behavior of the cords is being watched in the mirror. If the cords approximate and the entire length of the glottis closes even for a fraction of a second, and even if no sound is made, there is no organic adductor paralysis. If the cords approximate but there remains a triangular chink posteriorly (Fig. 379) there is an adductor paralysis involving the arytaenoideus only. If the glottis closes posteriorly but leaves an elliptical chink, and the cords lack flatness (edges rounded), the thyro arytaenoides are paralyzed ("tensor" paralysis). It is well to remember that in psychoneurotic aphonia from psychic trauma (*q v*) any adductive failure is never unilateral. Another important point in diagnosis is that organic adductor paralysis may be only part of the paralytic complex as mentioned under "Syndromes Associated with Laryngeal Paralysis." Etiologic localizing fixation or ankylosis of the crico-arytenoid joint must always be excluded before making a diagnosis of paralysis, this is readily done by the passive mobility test (*q v*). Diagnosis, which is always essential for proper treatment, is often a complex neurologic problem. A serologic test for syphilis will often solve the problem, if the reaction to the blood test is negative a spinal fluid test is indicated. If reactions to both tests are negative the tests should be repeated after a four-weeks' course of heavy doses—say about 30 grains (2 gm.) three times a day—of potassium iodide as a provocative. If the reaction is then positive it also serves as a *therapeutic diagnosis*, i.e., for treatment. Tuberculous interarytenoid infiltration must always be excluded, because, in some cases, it props apart the arytenoids, simulating an adductor paralysis. The presence of such an infiltration is readily determined by direct laryngoscopic palpation and the palpatory passive mobility test (*q v*).

Treatment.—This is chiefly general and depends upon the causative lesion. If the therapeutic diagnosis is syphilis the patient should have a suitably planned course of antisyphilitic treatment (see p. 20). If the adductor paralysis is due to cerebral concussion from a blow on the forehead, as in five of our cases, the usual general treatment for concussion is indicated. Toxic paralyses, if diphtheritic, require nuxvomica and iron, if metallic, as from lead, elimination of the source and systemic elimination of the metal are indicated. One local measure is sometimes urgently indicated. In

some cases adductor paralysis is associated with failure of the protective function (*q v*) of the larynx. Food and fluids may get into the larynx on swallowing, and oral secretions may trickle in. These patients need a frequently repeated laryngoscopic aspiration (*q v*).

Prognosis and Sequelae.—Prognosis is dependent upon the character of the lesion. In three of our cases of adductor paralysis there was complete, spontaneous recovery. The cause of the paralysis was cerebral concussion from blows on the calvarium. In a fourth case the patient died of other injuries. In the other case a complete paralysis followed the adductor paralysis (see Semon's rule). In another case, the cause of which was diphtheria, there was complete recovery. In our cases due to central or peripheral syphilis, motility did not return. The only local danger to life is from tracheobronchitis and pulmonary atelectasis, from inspiration of food, liquids, and oral secretions, due to impaired laryngeal protective closure and to failure of laryngeal tussive cooperation (*q v*). In cases in which the adductor paralysis is part of a complete paralysis the prognosis is that of the latter—bad as to recovery of function and unfavorable as to long survival of the patient. Apart from those of the causal condition the chief sequelae of permanent adductor paralysis are aphonia and chronic tracheobronchitis.

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EPIHEMERAL ADDUCTOR PARALYSIS

Ephemeral adductor paralysis is a transitory paralysis of the adductor muscles of the larynx due to sudden circulatory disturbance, usually anemia of the motor cortex. Synonyms in lay literature are "struck dumb," "knocked speechless," "speech bound," and "speech bereft."

The term, of course, does not apply to mental confusion in which a person does not say anything because he cannot decide what to say. No medical synonyms have been created because the laryngeal phases have not been recognized. *Transitory adductor paralysis* is an acceptable synonym. The condition is a relatively common occurrence.

Etiology and Pathology.—Ephemeral adductor paralysis is classed as, in a sense, organic because it is due to anemia of the anterior cerebral cortex. This vascular change with its sudden lowering of cerebral blood pressure suspends the emission of motor impulses from the executive centers in the lateral cortex (Fig 335). The pathologic physiology is the same as that in ordinary syncope, fainting, or swooning, of which the patient with ephemeral adductor paralysis may stop short. In other words, it occurs when the conditions causing syncope are, for that particular person and at that moment, insufficient in degree to cause unconsciousness. Swooning is usually silent. It is true that a person may scream from fright but if the spectacle is sufficiently appalling to cause fainting the passing into unconsciousness is always a silent interval, however brief. A person who faints seldom falls heavily because he has sufficient warning. Yet he makes no outcry. It is in this stage that the ephemeral adductor paralysis occurs and it is a stage of longer duration in those who do not quite reach the degree of cerebral anemia to cause unconsciousness. The accompanying pallor of the face is indicative of the cerebral anemia. The first cases in which we noted the adductor paralysis were in patients developing toxic symptoms, during operation in the sitting position, from local application of small amounts of cocaine. The circulatory failure in such cases is idiosyncratic and these conditions are entirely different from those of neurotoxic cocaine overdosage. When the facial pallor became extreme, it was noted that the patient, though he had not lost consciousness could not say "E-e-e" when requested, though quite familiar with such a request, and the cords were seen to remain in the abducted position. After the cerebral anemia was remedied by recumbent posture the patient recovered the power of adduction. Later corroboratory observations were made in many different kinds of cases of acute cerebral anemia, most of them on the border line of fainting. Some of them were medical freshmen first

witnessing an operation. Though emotion is concerned in producing the anemia, the anemia itself is a visceral condition associated with suspension of impulse production or of neurogenic transmission. This is a very different thing from the ordinary aphonia of psychic trauma (*qv*) in which there is no persisting cerebral anemia. In the latter class of cases the executive centers are unimpaired, there is a failure to use them that is a mental, not a physical, disability.

Laryngeal Appearances.—The cords are in the position of quiet respiration or less widely abducted. There is no apparent attempt at approximation when the patient is requested to say "E-e-e" no matter how well trained he may be in the procedure. All of the adductor muscles are obviously affected, the arytaenoides makes no attempt to approximate the arytenoid cartilages. The mucosa is pale. If the examination is by mirror the tongue usually must be seized in the mouth.

Symptoms.—The symptoms consist in aphonia, due to inability to approximate the vocal cords, in association with the symptoms of acute cerebral anemia, especially facial pallor, vertigo, dimness of vision, muscular weakness to the extent of inability to stand or sit up, impending loss of consciousness, and failing pulse.

Diagnosis.—The conclusive diagnosis, as with any other laryngeal condition, can be made only by laryngeal inspection, but the inferential diagnosis is easily made by the silence of the patient, who cannot answer any simple question and who makes no complaint notwithstanding the alarm and anxiety naturally present because of the dimness of vision, vertigo, nausea, and feeling of impending unconsciousness or "passing out," as one patient afterward explained it who said he wanted to speak but could not.

Treatment.—Local treatment of the larynx is obviously not indicated, though it is noteworthy that the pulling out of the tongue for mirror examination is not without some degree of resuscitating effect. For the cerebral anemia the best remedy is postural. The patient should be horizontal or inclined toward a low head. Reaction is prompt to inhalation of aromatic spirits of ammonia if aromatic spirits are not available, household ammonia may be used for inhalation, with proper caution, but never internally.

Prognosis.—The adductor paralysis in itself involves no danger to life, but, of course, it may accompany a fatal syncope. The voice is almost always recovered. In unstable persons the attack of ephemeral adductor paralysis may have as a sequela a psychoneurotic aphonia due to the psychic trauma (*q v*), with vocal fixation.

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BILATERAL MIDLINE ABDUCTOR PARALYSIS

Bilateral impairment of opening movements of the larynx due to impairment of innervation is known as bilateral midline abductor paralysis. Synonyms are *abductor paralysis*, *recurrent paralysis*, and *posticus paralysis*. *Paramesial paralysis* should be used to cover cases in which the cords are near the midline yet not quite phonetically approximated.

The paralysis of the abductor muscles on both sides is not uncommon. It is most frequent in regions where goiter is prevalent. It is not nearly so frequent as the unilateral type, but its accompanying dyspnea is so urgent that it is less often overlooked. In our experience at the Broncho-esophagologic Clinic it has been found to be less frequent in the proportion of 51 to 107 as compared to unilateral paralysis.

Etiology.—It is usually said that operation for goiter is the most frequent cause. Any goiter large enough to require removal has already imperiled the integrity of the inferior laryngeal neurons. Even a small goiter, if it has extended retrotracheally, has risked causing laryngeal paralysis. Our clinical experience has shown that an overlooked preoperative unilateral paralysis has been present in most cases of bilateral paralysis deemed postoperative.¹ Even when due to operation it is not to be construed as a cutting of the nerve. Healing and tumefaction and, later, cicatrization may cause paralysis on one or both sides (see also under "Reflected Appearances," p 527). Next to goiter, other

lesions in the long and circuitous pathways of the inferior laryngeal neurons are the direct causes (Fig 334). In fact, the chief predisposing cause may be said to be anatomic, because of the had routing of the inferior laryngeal nerve fibers which go down into the thoracic aperture and back up through the neck after turning around two large and often diseased vessels, the aorta on the left and the subclavian on the right. In this long course the lower neurons are exposed to compression or involvement by aneurysm, adenopathy, tumor, trauma, thickened pleura, tuberculosis, syphilis, and general surgical diseases of the head and neck. A review of our cases justifies the rough estimate that bilateral paralysis of the inferior laryngeal nerves would not occur more than half as often if these nerves were given off from the vagus at the level of their entrance into the larynx (Fig 334). Disease of the central nervous system, disseminated sclerosis, bulbar syringomyelia, and especially tabes are the most frequent central causes. These and others are referred to in the following paragraph, and also in connection with related paralyses and syndromes. Among toxic conditions causing abductor paralysis may be mentioned vegetal poisons such as alcohol and tobacco, metallic poisons such as lead, infections such as influenza, diphtheria, pneumonia, erysipelas, typhus, and hotulism. Age and sex have only indirect relation to cause of this form of paralysis. Our oldest patient was ninety-one years of age. We have seen it frequently in the newborn (*q v*) possibly due to slight trauma to the central or peripheral neurons during instrumental delivery.¹

Pathology and Localization.—The abductor muscles of the larynx are the pair of crico-arytaenoides postici. They are innervated by the abductor fibers in the respective inferior laryngeal nerves. The chief function of these muscles is to hold the glottis open for the entrance of air. When they are paralyzed the unopposed and powerful closing muscles shut the glottic airway. In their long course from the nuclei ambiguus (Fig 334) to their entrance into the larynx the laryngeal nerves, especially the inferior, are exposed to damage by pathologic processes in the surrounding tissues as well as within the nerves themselves (neuritis, toxic or other forms). In all this peripheral course the inferior laryngeal nerves, right and left, are on the respective sides of the midline tissues, consequently injury to one nerve will cause a uni-

lateral paralysis, injury to both nerves is necessary to produce bilateral peripheral paralysis (Fig. 334). This is true of all the nerve fibers (called "lower neurons") below the nuclei ambiguus. Above the nuclei, the corticobulbar fibers (called "upper neurons") decussate, and though the decussation is only partial it is sufficient to give motor control on both sides of the cerebral cortex (Fig. 334). Hence injury to the cortex or to the upper neurons of one side would not cause a unilateral paralysis, nor any paralysis at all, because the cortex and upper neurons of the sound side would still send the motor impulses to both nuclei ambiguus to activate both the respective right and left inferior laryngeal nerves originating therein. The cortical laryngeal motor areas (Fig. 334), right and left, are so widely separated that only a relatively enormous lesion, or an exactly located pair of bilateral lesions, could affect both sides. At or near the partial decussation, however, a smaller lesion can reach the upper neurons of both sides, and thus cause a bilateral motor paralysis of the larynx. Such a lesion, being above the nuclei ambiguus, would produce a *spastic* paralysis, whereas lesions affecting the lower neurons (those below the nuclei ambiguus) would cause a *flaccid* type of paralysis. The spastic (upper neuron) type of paralysis is usually overlooked because the larynx plays such a minor part in the manifestations of the terminal clinical features of such a serious case of cerebral disease. We have seen it in cases of pseudobulbar paralysis in which a paramesial paralysis later became midline as the result of spasticity. We believe spasticity explains the sequence of the midline position in cases of bilateral paramesial position and in at least some of the cases of bilateral incomplete paralysis.

The two nuclei ambiguus are not far apart, and the same is true of the respective laryngeal nerves that originate therein, in their courses to their emergence from the skull, encased in the respective vagi, through the jugular foramina. Hence any lesion in this intracranial region may cause either a unilateral or a bilateral paralysis. In one of our cases a chondroma caused first a unilateral, which was followed by a bilateral, paralysis. These clinical data show that an intracranial infranuclear lesion can be unilateral whereas central lesions are always bilateral. Outside the skull, in the jugular foraminal region (Figs. 334, 398), the vagi are

not too far apart for both to be affected by a large growth, but a paralyzing degree of involvement affecting both vagi would be fatal. Paralyzing compression of one vagus in this region sufficient to cause unilateral laryngeal paralysis is not infrequently seen in cases of tumor primarily in the region, or as an extension from the nasopharynx or sphenoid sinus. In considering central causes a distinction must be made between the bilateral laryngeal motor centers in the cortex, on the one hand, and the unilateral language (word) motor centers on the other hand (Figs. 334, 335). These associated one-sided word centers act upon the laryngeal motor centers through association fibers, and the peripheral effect in speech mechanism is bilateral. Pathologic tissue change in the language area does not produce laryngeal paralysis because the larynx does not make words (see under "Innervation in Relation to Speech"). As mentioned under "Hoarseness," it should always be borne in mind that syphilis, tuberculosis, hematoma, or tumors may produce laryngeal symptoms, not only as a local lesion in the larynx, but by a laryngeal paralysis due to such a lesion in the brain, the neck, or the thorax (Fig. 334). Other pathologic conditions associated with laryngeal paralysis may be listed as follows: neuritis of the laryngeal nerves, thickened pleura, pericardial effusion, dilatation of left auricle, diphtheria, trauma, abscess, aneurysm of the internal carotid, of the innominate or subclavian (right), of the aorta (left), adenopathy, disseminated sclerosis, glossolaryngopharyngeal paralysis, bulbar syringomyelia, tabes (bulbar form), poliomyelitis, encephalitis. Abductor paralysis forms a part of the group of paralyses in various syndromes (g.). The subject is further discussed under "Paralyses in Relation to the Esophagus."

The foregoing includes all the causes usually encountered clinically. To name all the remote possibilities would be to make a catalogic list of all subdermal structural diseases of the head, neck, and thorax. Such a list would create confusion for the laryngologist. With a clear conception of the innervation (Fig. 334), the muscles (Figs. 332, 333), the physiology (p. 421), and the laryngeal appearances herein described and illustrated the laryngeal phases of any case of laryngeal paralysis can be fitted into the general medical and surgical phases of any particular case.

Reflected Appearances—The characteristic of

midline bilateral abductor paralysis is that the larynx is paralyzed shut. Therefore phonation may be little affected, while the obstruction to inspiration is extreme. On phonation the glottis is seen to be almost closed, the cords are almost in contact and phonation is good, but on inspiration it is seen that the cords remain together and the patient has great difficulty and much stridor when drawing in air (Fig. 378). There are two exceptions to this difficulty. One is when the cords are at a different level the air may pass the cords one at a time like haffle plates, the edges of the cords being in line as seen by vertical mirror image, a considerable chunk is invisible. The other exception is in cases in which the obstructive paralysis has developed so slowly and lasted so long that the patient has learned to breathe slowly and quietly, never creating a great demand for air by exertion. Sudden onset usually causes asphyxia because the violent efforts to draw in air force the cords together (see Fig. 357).

Appearances on Direct Laryngoscopy.—Before doing a direct laryngoscopy the patient, if old enough to understand, should be told that the examination may shut off his air supply for a time, but that you will know when this occurs and will not keep it shut off too long, and further, that he must not be alarmed. If he is wearing a tracheostomic cannula he should be told that it will be occluded for a few moments now and then. The appearances through the anterior commissure laryngoscope are shown in Figure 378. The cords are in the same position as that of normal phonation, the difference is that at no time do they separate. When the patient is told to take a deep breath, or when the cannula (if one is worn) is occluded, the cords do not open to the inspiratory position. Through the anterior commissure laryngoscope the difference in level of the right and left cords, that is invisible in the mirror, is readily seen if present. Air should be admitted often during direct examination by passing the tube mouth through the glottis. The passive mobility test is always a part of the direct examination. The arytenoid eminences, which are tumbled forward, are easily moved backward, but they recoil. Flaccidity or spasticity can usually be differentiated by this test. The cricopharyngeal crescent (3, Fig. 369) should always be examined, it is almost or quite absent if the cricopharyngeus is paralyzed. In such cases the inferior constrictor is usually paralyzed also.

Symptoms.—Inspiratory dyspnea and stridorous breathing are the chief symptoms. The only exceptions are those mentioned in describing the reflective and direct appearances. In these exceptional cases the stridor and dyspnea will appear on slight exertion. If tracheotomy has already been done they will appear if the patient shuts off air by closing the orifice of the cannula with his finger. The voice may be weakened, slightly impaired in tone, and sometimes hoarse, especially on awakening, but usually it is so good as to mislead an inexperienced observer to doubt the presence of a paralysis. Noisy breathing during sleep, usually called "snoring" is the complaint of relatives.

Diagnosis.—Diagnosis is best considered in two parts, the laryngeal and the etiologic. The laryngeal diagnosis is based on the previously described reflective and direct appearances considered in contrast with the normal as well as with the appearances described in connection with other laryngeal paralyses. One step is essential to establish the diagnosis conclusively since fixation of both crico-arytenoid joints could give the same appearance of midline paralysis, fixation must be excluded. This is readily done by the mobility test. With the closed laryngeal forceps used as a probe through the direct laryngoscope the arytenoid cartilages when the posticus muscles are paralyzed, can be slid freely through their normal excursion, if there is no abnormality in the crico-arytenoid joint. Ankylosis is unmistakable to the trained touch, lesser degrees of fixation are easily determined. If the laterales are active there will be an elastic recoil.

Having thus established the presence of a bilateral midline paralysis it remains to determine the nature and location of the causative lesion. For this the following list of steps will be helpful. Pitfalls lurk when any one of these steps is omitted.

- 1 Anamnesis
- 2 General medical examination
- 3 Neurologic examination—central peripheral
- 4 Serologic tests—blood and spinal fluid
- 5 Roentgen ray examination—head, neck, thorax, swallowing function
- 6 Inspection of lips, mouth, tongue, fauces, palate, pharynx, nose, and nasopharynx, noting movements as well as tissues
- 7 Examination with laryngeal mirror
- 8 Direct laryngoscopy (first without anesthesia general or local, to determine presence or absence of sensory paralysis)
- 9 Passive mobility test

- 10 Palpation of laryngeal cartilages
- 11 Careful palpatory search for tenderness or a lesion throughout the course of all four laryngeal nerves
- 12 Bronchoscopic and esophagosopic search for lesions that might involve inferior laryngeal nerves

The differential diagnosis between a *spastic* and a *flaccid* paralysis in muscles of the larynx in some cases is a vastly more difficult problem than that in the muscles of an extremity. Not only are the laryngeal muscles partly concealed from view and relatively inaccessible for satisfactorily eliciting reflexes and clonus but some of them are normally concerned with producing hypertonicity and others with producing flaccidity. A pair of normal cricothyroides will modify the flaccidity of a pair of paralyzed postici, to some extent a normal arytaenoideus or a pair of normal thyro arytaenoides may be similarly confusing. There remain three means of differential diagnosis:

- 1 Observation with the mirror to note seriatim (a) the action or inaction of each individual muscle and (b) action or inaction of each group or part of group of muscles. For this it is necessary for the observer to have a perfect conception of the form, position and movement of each and every moving part of the laryngeal mechanism. This can be acquired by persistent prolonged intensive study of the normal, that is to say the unparalyzed, larynx.

- 2 Testing the mobility, flaccidity, spasticity and resistance to passive motion of each crico-arytenoid joint with the closed laryngeal forceps used as a probe through the direct laryngoscope. In doing this a start is made with a light local cocaine anesthesia and repeating the examination over and over again until the anesthesia has entirely disappeared. In this test the possibility of arthritic fixity of the joint is eliminated by observation as to any limitation of excursion apart from elastic muscular resistance (Fig. 378).

- 3 The flaccidity of the cricothyroid muscle can be detected by palpatory rocking of the thyroid cartilage on the cricoid. Here again training on the normal is necessary, hundreds of larynges must be palpated. This can be and should be done routinely at every laryngeal examination.

Complications.—The chief complication is asphyxia. Prior to maturity the constant indrawing may cause the funnel breast deformity with sinking in of the sternum and costal cartilages. Prolonged anoxemia and nocturnal attacks of severe dyspnea may greatly impair general health. Pulmonary complications may result from lack of normal pulmonary drainage because of failure of glottic cooperation in productive cough. In the tracheotomized patients, cicatricial stenosis is inevitable if the tracheostomic opening is not placed low, as it

should be. Unfortunately there are many such cases. ^{1 2} Bilateral abductor paralysis itself may be regarded as a complication of the basic disease—aneurysm, cardiopathy, neoplasm, or whatever it may be. The possibility of endocrine disturbances, manifested by myxedema, tetany, and mental disturbances, must be always in mind, especially in cases in which the paralysis has followed operation for goiter, but in other cases also. The acid base balance may be disturbed by the anoxemia, dyspnea, and carbonic acidosis incidental to varying degrees of respiratory obstruction in these cases.

Prophylaxis.—Among many other reasons, careful, thorough examination of the larynx in all cases of the slightest, even temporary, hoarseness is indicated for the early detection of a unilateral paralysis which so often is a forerunner of bilateral paralysis. In many such cases involvement of the nerve of the other side could be prevented. Our clinical records show many cases of bilateral paralysis in which the unilateral paralysis was overlooked, mostly cases of goiter and aneurysm. Possibly existing, potentially paralyzing intrathoracic conditions are an additional reason for including roentgen-ray examination in the periodical health examinations.

Treatment.—The primary indication is to prevent asphyxia. This usually requires tracheotomy, to which patients are reluctant to consent, unless dyspnea is extreme. It should be explained that a partial cork can be worn in the cannula, that it can be gaged to admit only sufficient supplementary air for daily comfort and quiet restful sleep, yet it is removable for occasions requiring more air. The valve cannula may be used. Asphyxia being forestalled, the next question is as to remedy of the laryngeal condition. The patient wishes to know if and when he is likely to get rid of the cannula. A cordal position of complete paralysis (Fig. 396), if it should supervene, would give more air with impaired voice, but it is too rare a sequela to be awaited, and operative production of it is uncertain and usually inadvisable. Nerve anastomosis so far has been unsuccessful. External operation dealing with the arytenoid joint has yielded satisfactory results in increased airway with a minimum of impairment of the voice. In a good percentage of the cases the patient has been decannulated and the fistula in the neck has been closed. In only a few such cases has it been necessary to perform a subsequent trache-

otomy for dyspnea, and this procedure can usually be avoided if the patient would be content with simply leaving out the cannula without a plastic closure, which would leave a small epithelialized fistula, for emergency use of a small cannula and pilot, as mentioned in connection with decannulation. In any operation dependent for its efficacy upon the dragging outward and rotation of the arytenoid eminence, it is of utmost importance to determine how far outward the arytenoid will be when healing is complete. If it is not far enough the patient will have dyspnea on slight exertion, if too far the breathing will be satisfactory but the voice will be husky and there will be an annoying sound of air waste with phonation. If the patient's occupation is active and requires physical exertion more than voice, the latter result would probably be most satisfactory, but if the patient needs a good voice rather than physical activity, the glottic chink should not be anchored too widely open for the cords to be pressed together, for phonation, by whatever degree of approximating power may remain or be vicariously developed. These elements are uncertain developments difficult to determine in advance. Before any procedure of this kind is undertaken the patient should be fully informed of the problem, namely, that the cords are close together and that is why his voice is so good, but because he cannot pull his paralyzed cords apart he cannot draw in enough air. The more airway given him the more his voice will be impaired. The decision, in the compromise between the two contingencies, should rest with the patient. If, for occupational or other reasons, he values his voice more than a very free airway, he probably must be content with limitation of active exertion. If, on the other hand, his occupation is physically active he should be content with some impairment of voice entailed by a wider separation of the cords. It should be further explained that the character of the operation does not permit precise results. No promises can be made except that as long as any air comes through his larynx he will not be without a voice. It is advisable, also, to tell the patient that the cannula cannot usually be dispensed with immediately after operation. In any operative procedure for relief of bilateral midline paralysis it is a serious responsibility to determine the elapsed time after which there is no hope of spontaneous recovery. Perhaps the best that can be done for the pa-

tient is to inform him that in case of no improvement whatever in airway after, say, six or nine months of midline position the hope of spontaneous recovery is remote. Unquestionably the results of external operation would be better if done immediately after the onset of the paralysis, yet the number of spontaneous recoveries is sufficient to render immediate operation inadvisable, though not unjustifiable.

Brien T. King was the pioneer in devising a satisfactory operation for bilateral midline paralysis. He has greatly improved his operation, and has supplied us, in the following article, with the full details of his latest technic and the plan upon which it is based.

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IMPROVED TECHNIC IN THE KING OPERATION FOR RELIEF OF BILATERAL VOCAL CORD PARALYSIS

Experience has shown that the results obtained in the operation for the relief of bilateral abductor cord paralysis were not always the same when the original technic, described in the March 4, 1939, issue of the Journal of the American Medical Association, was used. By a modification of that technic, described in the textbook, "Diseases and Injuries of the Larynx" by Chevalier Jackson and Chevalier L. Jackson,¹ an adequate airway was secured in a higher percentage of cases. Inequalities of voice have occurred following operation by either technic. Failure in securing a voice and airway of uniform standard is thought to be due more to inherent differences with which the surgeon is dealing than to alterations of technic. Human larynges vary in size and shape almost as much as human noses.

In the majority of patients, an adequate airway and a good voice were obtained. Consider-

able variation in each has occurred in the fifty-five patients studied after operation. The differences in voice range from an actual improvement in two instances to only a whisper in one. Most have some huskiness of voice.

As for airway, it may be said that an opening between the cords of less than 3 mm produces an inadequate airway and one of more than 5 mm will produce a poor voice although an excellent air supply. Patients learn automatically to vary and improve the airway but this is controllable only to a limited degree.

It can be stated with certainty that, following a bilateral operation for relief of paralysis of the vocal cords, the average voice will be less satisfactory than when operation is performed on one cord only. It is believed that most of the sound produced in the larynx following the operation is the result of vibrations of the undisturbed cord. Therefore the vibratory function must be preserved.

Contractures of the paralyzed vocal cords are not always of the same degree. Vocal cords vary considerably in length. The length of the cord in the male averages one and one-half times that of the cord in the female. It is easier to secure a good airway when dealing with a long cord. The larynx in the male is much larger than that in the female. The pyriform fossae of the larynx are wider in the male than those in the female. Therefore, during the operation a greater outward displacement of the arytenoid cartilage is permitted in the male than in the female. Also, the width of the fossae may vary considerably in individual members of the same sex. All factors must be considered in the course of an operation. Unless one has thoroughly studied the larynges of both sexes, it is not easy to make the necessary calculations.

Physiologically, it is by outward rotation of the arytenoid cartilage rather than by outward displacement that an adequate airway is produced. Postoperative study of these patients emphasizes this point. It was concluded that a technic that would displace the arytenoid cartilage outward in a state of partial rotation would be an improvement, as any failures in the operation were the result of insufficient airway.

At first a technic by which the arytenoid cartilage could be displaced outward and maintained in a position of partial rotation seemed difficult to achieve. However, two rather simple methods, herein described, were evolved. Both

are effective but one is technically easier to perform.

The omohyoid muscle is still transposed as a step in the operation but the value of this procedure is under question. It is now believed that the results obtained are chiefly due to the outward displacement and rotation of the arytenoid cartilage and the accompanying displacement of the corresponding vocal cord. Patients develop a definite capacity to widen and decrease the opening between the vocal cords. This function is thought to be due largely to the actions of the cricothyroid muscles supplemented by the suprahyoid group of muscles. Patients are also able, in some instances, to improve the quality of the voice by persistent effort. Repeated laryngeal examinations reveal that it is the undisturbed cord which develops the greater range of motion. This range seems to be due to tension and relaxation rather than abduction and adduction, though the result is somewhat the same.

Technic of Combined Tracheotomy and Laryngoplasty.—Tracheotomy is a necessary companion to laryngoplasty performed for the relief of paralysis of the vocal cords. About 25 per cent of the patients had been tracheotomized from one to fifteen years. In former articles it was stated that preliminary tracheotomy should always precede laryngoplasty and muscle transportation. I have recently performed tracheotomy and laryngoplasty at one sitting. The technic of the combined operations plus improvements will be described in the following paragraphs.

In patients with tracheotomy, preoperative sedation is recommended. My preference is for either $\frac{1}{4}$ grain (0.015 gm) of morphine or $\frac{1}{4}$ grain (0.020 gm) of pantopon combined with atropine 1/150th grain (0.0004 gm). To patients on whom tracheotomy is to be performed, be it a preliminary tracheotomy or combined with the plastic operation, any sort of sedation is absolutely contraindicated. This statement holds good as regards tracheotomy done under any and all circumstances.

The combined operation can be and has been performed under local anesthesia. However, this is less practicable than general anesthesia and renders the use of the laryngoscope during the operation difficult. Pentothal sodium given intravenously is highly satisfactory as an anesthetizing agent.

Tracheotomy.—Before performance of trache-

otomy, a syringe loaded with a solution of sodium pentothal is made ready. A needle is inserted into one of the veins at the elbow and the syringe connected to it by rubber tubing. When the tracheotomy operation has proceeded to a point where the trachea is opened and the cannula inserted, the pentothal solution is injected into the vein, and the procedures from thereon are continued under general anesthesia.

The patient's neck is prepared by any of the accepted skin cleansing technics. My preference is soap and water, followed by alcohol, ether, and merthiolate. One per cent of novocaine is then injected, infiltrating the tracheotomy area. If the patient has previously had a thyroidectomy, I prefer a transverse incision for the tracheotomy rather than a midline incision. Tracheotomy wounds are never aseptic. Transverse incision will be further removed from the laryngoplasty incision than if tracheotomy is done using a midline incision.

An incision $1\frac{1}{2}$ to 2 inches long is made. Skin flaps are dissected above and below just as is commonly done in goiter operations. The fascia is opened by an up and down incision and the muscles separated. This usually exposes the trachea as in most instances the isthmus of the thyroid gland will have been removed. The trachea is opened longitudinally. The opening should begin about $\frac{1}{2}$ inch below the level of the transverse incision in the skin. Otherwise when the skin is closed the upper flap will exert pressure and leverage on the cannula with the possibility of pressure decubitus on the posterior wall of the trachea at the lower end of the cannula. One or two chromic catgut sutures is taken in the muscles above and one below the cannula. The skin is closed with interrupted silk sutures. If the opening in the trachea is made at the proper level the skin may be closed snugly around the cannula with no tendency for the upper flap to override. A no. 5 cannula for women and no. 5 or no. 6 for men should be used.

After the tracheotomy is completed a small catheter attached to a suction tube is inserted into the opening for the purpose of removing blood or clots which may have accumulated in the trachea or bronchi. At this point a redraping of the patient is necessary. A woven wire tea strainer is placed over the tracheotomy cannula, the mesh of the strainer having been at one point spread by a pointed hemostat. Into this opening a catheter connected to an oxygen tank is inserted. Over the strainer and tracheotomy field a sterile towel is spread, the edge of which is placed just above the rim of the strainer. Over the towel is placed a sterile 2 foot square of rubber sheeting or oiled silk. Both are fastened snugly by means of skin clips to the skin above the strainer and transverse incision. This is done to prevent expired air from the tube blowing over the operative field.

Laryngoplasty—The skin above the tracheotomy area is repainted. The operating team

changes gloves and gowns and the patient is redraped with sterile drapes. A new set of sterile instruments is used.

An incision $2\frac{1}{2}$ inches or 3 inches long is made over the lateral border of the larynx or thyroid cartilage. It should be parallel to and $\frac{1}{2}$ to $\frac{3}{4}$ inch in front of the anterior border of the sternomastoid muscle. To a right handed surgeon the left side of the neck will offer an easier approach. The skin and platysma muscle are incised. The superficial layer of the deep fascia and the tissues lying between the sternomastoid muscle and the lateral border of the thyroid cartilage muscles are opened. The incision exposes the anterior belly of the omohyoid muscle. Now its inner border and posterior surface are freed. The outer border is avoided except for a distance of $\frac{1}{2}$ inch nearest its attachment to the hyoid bone. The muscle is too long to exert traction on the arytenoid cartilage unless shortened to the proper length. Its tendinous end cannot be used for attachment to the arytenoid cartilage because of excessive length of the muscle. The sheath of the muscle is so thin that it offers little support to sutures. This problem is met by ligating the muscle belly with chromic catgut about $\frac{1}{2}$ inch below the hyoid bone. It is divided distally to the ligature. The ligature creates a cuff behind which sutures will hold. When the sternohyoid and sternothyroid muscles are retracted forward the fibers of the inferior constrictor of the pharynx appear. These fibers are attached to the border and outer plate of the thyroid cartilage and must be cut. The pharyngeal mucosa is then exposed. It contains neither longitudinal nor circular fibers is quite thin and care must be exercised in separating it from the border of the thyroid cartilage pyramidal fossa and the posterior surfaces of the cricoid and arytenoid cartilages.

There is a space of about $1\frac{1}{2}$ inches along the outer border of the thyroid cartilage in which no important structure is to be encountered. It extends from the superior cornu to the inferior cornu. At the superior cornu there is danger of injury to the internal division of the superior laryngeal nerve. At the inferior cornu there is danger of injury to the external division of the superior laryngeal nerve.

When the posterior surface of the cricoid cartilage is exposed the atrophic fibers of the crico-arytenoideus posticus are brought into view. This is the best landmark for the moment. The upper rim of the cricoid cartilage is then felt and the palpating finger slid outward on this rim or border. In the unparalyzed larynx it is easy to palpate the arytenoid cartilage, particularly its muscular process. This is not true in the paralyzed larynx, however, because the arytenoid cartilage is drawn forward by contracture into a horizontal rather than the normal perpendicular position. If one observes the fibers of the posticus muscle and cuts them with a small sharp scalpel at a point about $\frac{1}{4}$ inch from the upper end the crico-arytenoid joint will be opened. This joint like any joint that has been immobilized for months or years will be found in a state of ankylosis. In order to mobilize it the joint capsule must be divided completely. There is an internal lateral ligament to the joint which dips over the rim of the cricoid and is attached to the anterior surface of the cricoid. The ligament is small but strong and its division is important.

The fibers of the interarytenoid muscle are then divided close to the arytenoid cartilage. A small skin hook is passed around the arytenoid underneath the laryngeal mucosa. By this means one can gauge the amount of mobility that is established. It is desirable to cut a few fibers of the crico arytaenoideus lateralis. By so doing a greater degree of mobility is secured. Then for a successful displacement of the cartilage almost complete disarticulation is essential. Maintenance of the proper degree of displacement and rotation must be the object of the next procedure.

Because of inequalities in the width of air ways secured by the technic originally described two modifications of it have been

notch in the border of the thyroid cartilage into which the arytenoid was displaced and fixed. This technic produced a greater outward displacement of the arytenoid and uniformly secured a wider airway. In one patient in whom it was used an infection developed in the wound which resulted in the infection invading the thyroid cartilage with a consequent small draining fistula which required nearly one year to heal. The functional result was not influenced by the infection.

As infections have occurred in a higher percentage of these operations than is ordinarily

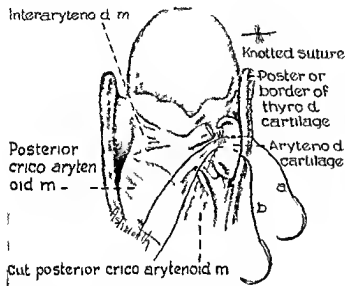


Fig 380



Fig 381

Fig 380—This is an excellent illustration of just what is desired to be accomplished in the operation. The cricoarytenoid and interarytenoid muscles have been divided. The cricoarytenoid joint has been thoroughly disarticulated (note joint facets). The arytenoid cartilage is displaced outward in a state of partial rotation. Note that the arytenoid cartilage is in contact with the outer border of the thyroid cartilage. This is the position which has given the highest percentage of good results. In passing the needle around the arytenoid cartilage, caution should be exercised. Hug closely to the cartilage as it is easy to perforate the laryngeal mucosa and thus infect the wound (Davis and Geck have manufactured a special atraumatic needle and suture for this purpose. It is sold under the number P271.)

Fig 381—This illustration shows the completed operation with the arytenoid cartilage fastened in outward position and the omohyoid muscle fixed to its muscular process. The wound is now ready for closure, which completes the operation.

made. In the original procedure two sutures were passed either through or around the arytenoid cartilage. One of these was passed through the posterior border of the thyroid cartilage and tied tightly. The other was used to fasten the omohyoid muscle to the arytenoid. Efforts to pass a needle through the cartilage were abandoned because of the frequency with which the cartilage was fractured. The first real change in technic was described in the textbook by Jackson and Jackson.¹ It consisted of cutting a

notch in the border of the thyroid cartilage into which the arytenoid was displaced and fixed. This technic produced a greater outward displacement of the arytenoid and uniformly secured a wider airway. In one patient in whom it was used an infection developed in the wound which resulted in the infection invading the thyroid cartilage with a consequent small draining fistula which required nearly one year to heal. The functional result was not influenced by the infection.

As infections have occurred in a higher percentage of these operations than is ordinarily experienced in aseptic operations and as infections of cartilaginous tissues are notoriously difficult to heal, it seemed desirable to devise a technic which would be less subject to this hazard.

From the beginning it was realized that the operation failed in one detail to conform with the normal physiology of the larynx. The normal vocal cords are opened to a larger degree by outward rotation of the arytenoid cartilages than by outward displacement. A method of

technic securing both outward rotation and displacement of the arytenoid cartilage seemed desirable. Such a technic has been devised and in all instances where it has been used an adequate airway and a usable voice have been secured

and do a more thorough disarticulation of the crico-arytenoid joint. The second suture is threaded with a needle on each end and both ends passed through the omohyoid muscle where they are tied. The fibers of the inferior constrictor muscle are then sutured, the fascia and skin closed.

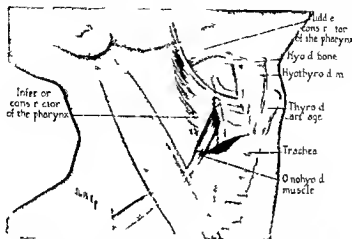


Fig. 382—Relief drawing which illustrates the new position of the omohyoid muscle and the sutured fibers of the inferior constrictor of the pharynx.

When the arytenoid cartilage has been thoroughly mobilized and the interarytenoid muscle divided along with some fibers of the crico-arytaenoides lateralis a hook is passed around the cartilage. This greatly aids the surgeon in passing a needle around the cartilage. The needle should closely hug the cartilage and not enter the laryngeal cavity. A full curved rather strong trocar needle threaded with no. 0 chromic catgut is used. The diameter of the curve of the needle should be about $\frac{1}{4}$ inch. The needle should always be passed from the mesial side outward.

The arytenoid cartilage on cross section is somewhat triangular in shape. Two sutures only are passed around the cartilage. One is for rotating and displacing the cartilage, the other for fastening the omohyoid muscle. A few inches of the tail of the upper of the two sutures are cut off and folded three times. This makes 8 strands around these 8 strands the same suture is tied. The ends of the 8 strands are cut off with scissors leaving a bundle $\frac{1}{4}$ inch in length. The purpose of this is to create a large knot which will hang in the tissues at the anterior edge of the arytenoid cartilage. The needle is then passed through the thyroid cartilage near the border. When the needle is pulled the arytenoid cartilage is displaced outward in a moderate degree of rotation (see Figs 380-381). At this point the wound is covered with sterile gauze and a sterile towel thrown over the field. A laryngoscope is then passed and while the assistant makes traction on first one and then the other end of the knotted suture the position of the cord and width of airway are thus observed. It is well for the surgeon to have familiarized himself with the larynx before operation and also to observe through the laryngoscope what has been accomplished by it. If the laryngoscopic examination shows a satisfactory airway, the suture is tied with greater traction on the needle end. If there is doubt about having produced an adequate airway it is advisable to go back



Fig. 383—This is a drawing of the actual appearance of the cadaver's larynx following operation. It does not truly represent the appearance of the paralyzed larynx. In the paralyzed larynx the right cord will be seen with the vocal band in a perfectly straight line and occupying a midline position. The left cord will also be in a straight line but in a position of outward displacement, but with less airway than is shown in this illustration. It is not desirable to produce an airway as great as is portrayed in the illustration. The cords in the paralyzed larynx are more rigid and therefore more difficult to displace than in the unembalmed cadaver's larynx. One should not expect to visualize a similar position of the cords to that shown in this illustration.

This operation was originally conceived on the theory that the omohyoid muscle would re-

place the function of the paralyzed crico-arytaenoides posticus. That theory has not been well sustained. It is now believed that the transposed muscle plays a role of minor importance and that the results obtained are primarily the result of outward rotation and displacement of the arytenoid cartilage and its corresponding vocal cord.

Instead of using the knotted suture for securing rotation and displacement, the same result may be obtained by passing one needle twice around the arytenoid. Pulling on the needle end of the suture produces a rotating effect.

The laryngeal ensemble is a complicated one, it is too lightly and glibly passed over in the dissecting room and in anatomies. To appreciate its physiology and disturbances in function, and particularly to correct any critical malfunction, will demand a detailed dissecting-room knowledge of its architecture, innervation, and musculature.

The operation should not be undertaken on a living subject but should first be performed on an embalmed cadaver.

BRIEN T. KING

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THE KELLY ARYTENOIDECTOMY FOR RELIEF OF BILATERAL LARYNGEAL PARALYSIS

Joseph D. Kelly modified the Brien King operation by excising the arytenoid cartilage through a window made in the thyroid cartilage.¹ The only special instruments required are a single and a double hook (Fig. 384). To the instruments required for laryngo-fissure may be added a small rongeur forceps for nipping off the edge of the cartilage in forming the window in the thyroid cartilage. If the patient is not already wearing a cannula, tracheotomy (*q.v.*) is done under local anesthesia, and a proper cannula is inserted. For the arytenoidectomy ether is given through the tracheotomic cannula until the patient is relaxed, then a Flagg rubber

intratracheal tube, size 5 to 7 mm., is inserted for continuation of the operation and for the support it gives to the arytenoid cartilage that is to be removed. The tracheotomic cannula is removed. The incision is made as shown in Figure 385. It is deepened to expose the sterno-

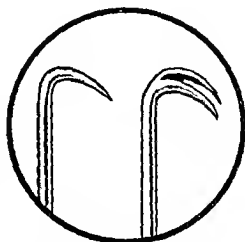


Fig. 384—Special hooks for arytenoidectomy

thyroid, sternohyoid, omohyoid, and the thyrohyoid muscles which are separated, clamped, and cut as in thyroidectomy. The thyroid cartilage is exposed (Fig. 386). In this a square window, 1 to 1.5 cm. in size, is made by incision

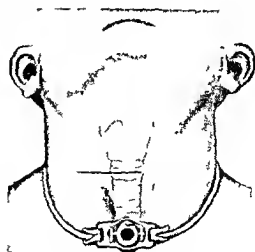


Fig. 385—Incision for arytenoidectomy in the Kelly operation

with a small knife, care being taken not to go through the inner perichondrium. The cartilage is removed with the small rongeur, then the inner perichondrium is cut away with scissors to expose the muscular tissue over the arytenoid (Fig. 387). The crico-arytenoid joint is exposed

by careful dissection through the lateral crico arytenoid and the thyro arytenoid muscles. The ligamentous capsule of the joint is incised with scissors and the arytenoid cartilage is held with

wound is closed a cigaret drain is inserted through a puncture wound made in the skin over the thyroid cartilage and dressings are applied. The intratracheal tube is removed and

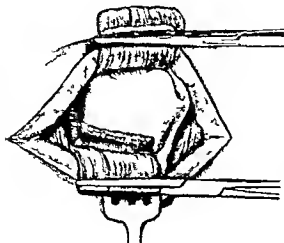


Fig 386

Fig 386—Exposure of the thyroid cartilage by severing and turning outward the cut ends of the cervical muscles. Fig 387—Window in the thyroid cartilage exposing the endolaryngeal muscles: the crico-arytaenoideus lateralis and the thyro-arytaenoideus. Through the fibers of these muscles the arytenoid cartilage is to be removed.

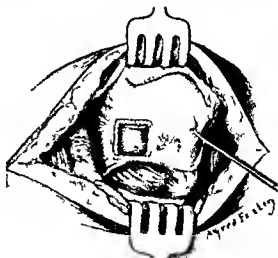


Fig 387

the double hook while it is carefully dissected free (Fig 388). The vocal process at the posterior end of the vocal cord is identified and is last to be detached. Care is necessary to avoid

the tracheostomic cannula is replaced. A feeding tube is inserted through the nose and allowed to remain for five days. Healing is complete within a week or ten days. The reflected appear

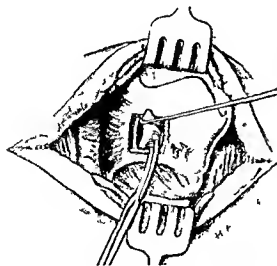


Fig 388

Fig 388—Freeing and delivering the arytenoid cartilage through the muscles.



Fig 389

Fig 389—Result two months after the Kelly operation.

either perforating the mucosa or cutting the cartilage. As the arytenoid is freed it is worked upward by gentle traction on the double hook and delivered through the window. The neck

ance at the end of two months is shown in Figure 389.

Edward S. Wright's Modification of the Kelly Operation—To secure a wider glottic space

posteriorly, Wright advocates that in all cases after the arytenoid cartilage is delivered the extreme posterior end of the vocal cord be

McCall and Gardiner Modification of the Kelly Operation³—These authors, instead of removing the arytenoid cartilage anchor it to



Fig 390—McCall Gardiner modification of Kelly operation for relief of dyspnea in bilateral midline laryngeal paralysis. The external operator has exposed the right ala of the thyroid cartilage (From McCall Julius W, and Gardiner Frederick S. A Simplified Operation for Bilateral Abductor Paralysis. *Laryngoscope* 53:307, 1943.)

Tracheal cannula



Transillumination room darkened

Fig 391—The laryngoscopist has covered the right arytenoid cartilage with the distal end of the anterior commissure laryngoscope and has pushed the arytenoid against the inside of the ala. In the darkened room the transillumination indicates the best place for cutting the window in the thyroid wing. This photograph was made by time exposure and it is not retouched (From McCall Julius W and Gardiner Frederick S. A Simplified Operation for Bilateral Abductor Paralysis. *Laryngoscope* 53:307, 1943.)

sutured to the external perichondrium at the lower margin of the window with no 000 chromic catgut so as to obtain about 6 or 7 mm width of glottis posteriorly.²

the window in the thyroid ala, and use the direct laryngoscope (1) to determine by transillumination the exact location at which to make the window, (2) for aid in presentation and fixation

of the arytenoid, (3) for determination of the desired width of the glottic chink posteriorly. Their technic requires two operators—one for the external operation, the other for the endoscopic work. As soon as the external operator has exposed the thyroid ala (Fig 390) the endoscopist covers the arytenoid with the tube mouth and presses it outwardly against the exposed thyroid ala. The room being darkened, the transillumination through the thyroid wing shows just where the window should be made (Fig 391). After the window is made the anterior commissure laryngoscope is reintroduced by the endoscopist. The operator uses a small full-curved needle to pass a fixation suture which is tied to the anterior part of the ary-

question as to whether or not there is any endocrine disturbance, especially in patients who have had a thyroidectomy. Usually, at the end of two weeks, the patient begins subjectively to realize a decided improvement in his airway and, naturally, wishes to know how soon he can abandon his cannula. This involves a decision that must be determined by circumstances in the particular case. The first step is to check on the size of cannula to be sure there is ample hy-passageway for air, as there always should be with a properly fitted cannula.³ If deemed necessary the next smaller size may be substituted (see "Decannulation" under "Tracheotomy"). The patient may then make frequent tests of airway through the larynx by occluding the

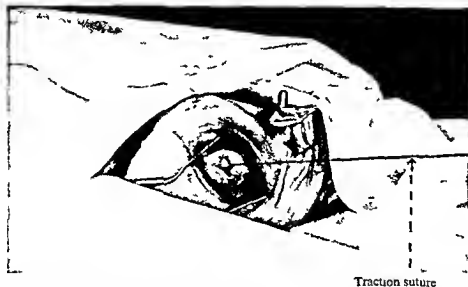


Fig 392—The window having been made, and the crico-arytenoid joint having been disarticulated, the freed arytenoid cartilage, by means of a suture is drawn to the window and anchored there. (From McCall, Julius W., and Gardiner, Frederick S. A Simplified Operation for Bilateral Abductor Paralysis, *Laryngoscope* 53: 307, 1943.)

enoid cartilage. The interarytenoid muscle is severed, and the crico-arytenoid joint is disarticulated so that the arytenoid cartilage can be drawn by the suture to the window (Fig 392) where it is anchored with 01 chromic catgut in a position that will give the desired amount of widening to the glottic chink posteriorly.⁴

Decannulation after Operations for Relief of Bilateral Midline Paralysis—Though the mucosa has not been perforated at operation there is the endolaryngeal edema naturally to be expected because of the proximity of the operative field. It interferes temporarily with swallowing as well as breathing through the larynx. This usually subsides within ten days. Any great delay in its subsidence beyond this time raises a

cannular orifice with his finger. When he is certain he thus gets plenty of air he is ready for test by corking the cannula (*q v*). When he can sleep comfortably night after night for a month or two he may be decannulated if he is in close touch with medical advisers. He should not go far from observation for a further period of a few months. If he has worn a cannula for many months the tracheostomic fistula, in most cases, will have been so thoroughly epithelialized with dermal epithelium that it will not spontaneously close. This is the patient's safeguard and it should not be closed by a plastic procedure (*q v*) for a number of months longer. During this period, in most cases, a cannula of small diameter (about 3 mm) with pilot, and

well greased, could be insinuated in the fistula for relief of dyspnea by giving supplementary air, in case of emergency. It is seldom advisable, unless the patient is closely in touch with medical aid, to close the tracheotomic fistula until the patient has passed through a few attacks of acute infection of the respiratory passages, such as are epidemic every winter in the north temperate zone. Such an attack may precipitate extreme dyspnea that may require a tracheotomy for at least temporary relief if no old fistula exists. There is usually very little leakage of mucus from an old fistula. Another contingency requiring periodical observation and care is endolaryngeal edema from endocrine disturbance in cases in which the bilateral paralysis has followed a gouter operation. As pointed out by Holinger,⁵ control therapy is best determined by clinical and hematologic response because the dyspnea, anoxemia, and other factors render the control by the basal metabolic rate inaccurate.

Prognosis and Sequela.—Bilateral midline paralysis is dangerous to life because of asphyxia for want of a tracheotomy. If asphyxia be prevented, death from laryngeal condition is relatively rare, and tracheotomy adds almost no risk. *Functionally* the prognosis as to the protective, tussive, and phonatory duties of the larynx is not bad. *The respiratory function is* hindered and, unless care be taken in gauging the by pass in the cork or the size of the valve cannula, circulatory balance may be impaired. In an extremely small percentage of the cases an incomplete or a complete paralytic position of the cords (*qv*) may ensue. The incomplete paralytic position improves the airway but impairs the voice; the complete position improves the airway much more, but makes the voice much worse. The speech is intelligible, but the voice has a stage whisper quality and there is added an annoying sound of air waste. Spontaneous recovery from a midline paralysis occurs in a small percentage of the cases in which the primary causative factor has been removed—less than 7 per cent in our experience. The prognosis as to airway and voice after operation for decannulation cannot yet be stated in percentages for lack of a large enough number of cases, but in a general way it may be said the prognosis is favorable.

The sequela most to be dreaded is cicatricial subglottic stenosis from the error of high tracheotomy.

CHEVALIER JACKSON

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BILATERAL INCOMPLETE PARALYSIS OF THE LARYNX

Paralysis of all the laryngeal muscles except the arytaenoides is known as bilateral incomplete paralysis of the larynx. This form of paralysis in the literature has been sometimes loosely called "cadaveric" and at other times "complete." Both of these terms have been incorrectly used to describe cases in which the paralysis obviously affected only the thyroarytaenoides. The worst error often appearing in the literature has been to call this form of paralysis "complete" when the accompanying illustrations and descriptions show it to be incomplete, they show the arytaenoides was not paralyzed. Obviously, in using the term "cadaveric," the various authors were not talking about the same thing. To clarify the confusion in the use of the term "cadaveric" we have gone over our old records and sketches, from which we can say that in about 75 per cent of the cadavers in anatomic rooms the position was relaxed, paraboloid, and similar to what many writers call "cadaveric," but which is really the position of incomplete paralysis. In the remainder it was various, in some instances, the position was paramesial, in others in various degrees of abduction, not always symmetrical. In our sketches of postmortuary larynges prior to the onset of rigor mortis, the position of the

cords was that often called "cadaveric" in only about 60 per cent of the cases. During rigor mortis, in some cases, we have found the cords of the normal larynx in the midline position with glottis almost closed by approximation of the cords as in phonation. After rigor mortis had passed off the cords, in some instances, were in a relaxed position approximating that usually called "cadaveric," but this was not the case in all. After the onset of decomposition, and in some instances before the onset, the position of complete paralysis appeared.

For the sake of accuracy it would seem best to drop the term "cadaveric" and substitute the word "incomplete" for the numerous cases in which the arytaenoides is not paralyzed. This would leave the term "complete" for proper application to cases in which the muscles—abductor, "tensor," and adductor, including the arytenoides—are all paralyzed.

Bilateral incomplete paralysis in our experience has been less frequent than midline paralysis in the proportion of 117 to 271.

Etiology and Pathology.—In the foregoing pages we have considered paralysis of the adductor muscles, the so-called "tensor muscles," and the "abductor muscles." All of the muscles in these three groups except the arytaenoides may be paralyzed and yet the vocal cords may remain near the median line (paramesial paralysis). There is in addition to the direct innervation of these muscular groups another form of innervation, presumably a reflex arc, called "tonus" (see under "Physiology of the Larynx"). When this tonus is also gone the cords lie almost or quite motionless in the position into which normal cords sometimes fall after death, hence the loosely used name "cadaveric paralysis." When this condition is reached progressively, during life, as the result of an organic lesion, usually the abductors are first affected, next the "tensors," then the adductors, and last the tonus. Semon felt so sure that this sequence always was followed that he regarded it as a law, and it is usually called "Semon's law." This sequence does occur, we have observed it, there are exceptions, however, that render it better to call it *Semon's rule*.¹ Incomplete (called "cadaveric" by Semon and others) paralysis, however, is not always reached progressively. We have seen it follow gonorrheal operations, in some cases as a transitory and in other cases as a permanent condition. In both these types it would be logical to assume that it

was due to peripheral operative injury if it were not for another clinical fact, namely, that we have seen it follow as a transitory condition after operations in relatively remote regions—the abdomen in two instances, for example. In this last class of cases, it seems reasonable to assume, there were intracranial disturbances, probably circulatory. As mentioned elsewhere (see under "Adductor Paralysis of the Larynx") we have seen a bilateral incomplete paralysis follow an adductor paralysis due to a blow on the calvarium. This is a reversal of the order in Semon's rule which places the adductors last to be affected. In other cases we have seen the so-called "internal tensors" (the thyro-arytaenoides internus) first affected, then the postici, and finally the adductors. Exceptions are not so numerous, however, as to nullify the rule though they are too many for a law. There seems ample clinical evidence to show a greater vulnerability of the posticus as a clinical fact. There may be more than one reason for it but the most plausible one is that the closing muscles of the larynx are much older in evolutionary progress than the opening muscles or the tensors.²

Anesthesia of the mucosa in some of the cases of bilateral incomplete paralysis would seem to indicate that loss of both the superior laryngeal nerves were concerned in the loss of the tonus arc, but the anesthesia is not present in all the cases.

Reflected Appearances.—In bilateral incomplete paralysis the vocal cords lie motionless midway between the position of deep respiration and that of phonation. The edges of both cords are symmetrically concave. The cords seem narrowed because they have sagged outward under the overhang of the respective ventricular bands (Fig. 339). The glottic chink is almost ellipsoidal in outline and alters little if at all during inspiration, expiration, or phonation (Fig. 396).

Appearances on Direct Laryngoscopy.—On the approach of the distal end of the anterior commissure laryngoscope, passed without anesthesia (general or local), the appearances are striking and unmistakable to the experienced examiner, especially in relation to movement. The glottis does not close during reflex cough. The cords may flutter slightly but they do not approximate at any time, nor do they open widely on deep inspiration. The contrast with the sudden opening and snapping shut of the

normal glottis under direct laryngoscopy done without anesthesia (general or local) is very striking. It is by this kind of examination that the distinguishing feature of incomplete paralysis is revealed, namely, more or less power of movement remaining in the arytaenoides (Fig. 393). Some of the patients with incomplete paralysis may come in wearing a tracheotomic cannula. In such cases the cannula should be momentarily occluded by the finger of a nurse, or a full cork may be worn during the examination.

Symptoms.—The chief symptoms are a weak husky voice and a fricative sound of air waste when the voice is forced which is annoyingly loud when the mouth is open. If the patient is a nocturnal mouth breather, the family are likely to complain about the noise they misinterpret as "snoring." There is a wheezy cough. When accumulating secretions are present in the tracheobronchial tree, as they usually are, the casual observer would make an intuitive diagnosis of asthma, in most cases of long duration this diagnosis is reported as having been made. The patient may complain of shortness of breath, but there are no signs of obstructive laryngeal dyspnea (*q v*) except on exertion. One complaint is common, namely, difficulty in "getting up secretion."

Diagnosis.—The image in the mirror, at rest and on phonation (Fig. 396), as described in a preceding paragraph, is conclusive. No physician familiar with the normal laryngeal image could mistake a well developed case with typical appearances. Usually these patients tolerate mirror examination well. Direct appearances are unmistakable. The diagnosis and localization of the causative lesion is the same as given for midline paralysis. The lesion in incomplete paralysis is usually above the giving off of all four laryngeal nerves, that is to say, in the laryngeal neurons in the vagus or before entering it (Fig. 334), but this is not always the case. The possibility of tuberculosis must always be excluded when the vocal cords are in the position of incomplete paralysis. In these cases, in addition to feebleness of muscles, complete adduction is mechanically prevented by tuberculous infiltration of the interarytenoid space. The presence of slight degrees of such infiltration can be determined only by direct laryngoscopy, using the closed laryngeal forceps for palpation and for the "passive mobility test" (*q v*).¹

Treatment.—Unlike midline paralysis, the incomplete form presents no urgent dyspnea. Direct treatment of the larynx is not usually called for. The causative lesion supplies the indications for treatment. Syphilis is one of the most frequent causes and should always be searched for promptly and thoroughly. If found it should be treated as elsewhere herein suggested. If the cause of a laryngeal paralysis has been eliminated, or if no cause can be found, spontaneous cure should be awaited. Two things are essential while waiting. First, the patient, by medical care and management, should be kept in best possible state of health, fatigue should be avoided, and an excess of mixed vitamins should be given. Second, the possibility of the cords assuming the midline position should be kept in mind because if this should happen, though the voice would improve, dyspnea would develop. The patient should not go anywhere remote from prompt tracheotomic aid. The midline position, when it occurs in these cases develops gradually, as a rule, but there are exceptions.

Prognosis.—In some cases of incomplete paralysis there is a spontaneous perfect recovery. In other cases the incomplete position is followed by a midline position, with improvement in voice but also with severe dyspnea. Asphyxia may occur for lack of a tracheotomy. In other cases the arytaenoides becomes paralyzed and the paralysis becomes complete (*q v*).

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COMPLETE PARALYSIS OF THE LARYNX

Complete paralysis of the larynx means paralysis of all the muscles of the larynx. This form of paralysis is often called "cadaveric" in the literature, but, as this term is also often ap-

plied to obviously incomplete paralysis, accuracy requires that the term "cadaveric" be dropped. Another good reason for dropping it is that the cordal position to which it is often applied, namely, midway between full abduction and close approximation, is often followed by the midline position, whereas complete paralysis never is. It always persists for life.

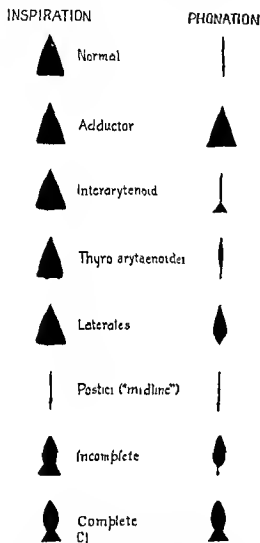


Fig. 393.—Glottic silhouettes in cases of bilateral laryngeal paralysis, contrasted with the normal

Complete paralysis is less frequently seen than either midline or incomplete paralysis.

Etiology and Pathology.—The pathologic condition present is, obviously, the complete destruction of the power to generate or conduct motor impulses that normally innervate the larynx. Normal innervation (*qv*) includes not only abduction, adduction, and "tension," but also a reflex tonus. When all these are gone the

cords and arytenoids fall into the position shown at the right in Figure 396. From disuse the muscles shrink and all the endolaryngeal tissues become atrophic. The chief causative lesion is usually above the giving off of the superior laryngeal nerve from the vagus, but not always. We have seen many typical cases due to aneurysm of the aorta, and to cervical as well as high intrathoracic tumors. In these cases there were probably secondary changes in the vagal neurons above the giving off of the superior laryngeal nerves. Complete paralysis is always bilateral because the arytenoid muscle cannot be unilaterally paralyzed. Not only can the muscle be innervated by either the right or the left nerve alone, but if the muscle contracts at all it pulls on both sides equally. In the patients sent in with a diagnosis of unilateral complete paralysis we have found the one-sided appearance to be due to ankylosis of one crico-arytenoid joint.

Reflected Appearances.—The glottic aperture is made up of an ellipsoid anterior to a triangle (Figs. 393, 396). The tissues are atrophied, the muscular tissues shrunk, the ventricular bands have lost bulk, the vocal cords are thin, translucent, weblike, with branching vessels conspicuous. The whole image is best described as "wooden," compared to the living moving larynx. The cords and arytenoid cartilages are seen to be immobile, they may be tilted forward. On attempted phonation the cords are immobile, there is not the slightest movement on attempted phonation, the air comes through the unchanging glottis with a loud hissing sound.

Appearances on Direct Laryngoscopy.—When the distal end of the anterior commissure laryngoscope approaches the glottis without the use of any anesthetic, general or local, the appearance is unmistakable. The absence of the usual sudden opening and snapping shut is striking. There may be a sensory paralysis of the laryngeal mucosa, but some reflex cough is excited by the extrinsic laryngoscopic tubal contact and it is noted that the cords do not approximate on coughing, instead there is a hollow wheezing hissing sound that is characteristic. The glottic silhouette is shown in Figure 393.

Symptoms.—The voice is rough and husky with a typical rasping sound and air waste, there is a monotonous lack of change of pitch. More or less cough is usually present and it

has a wheezy asthmatoïd sound. Expectoration is difficult and tracheobronchial secretions are annoying to the patient and cause a sound disagreeable to others. Sleep is noisy. Although there is no laryngeal obstruction, the glottic chink, being amply large, the patient may complain of air hunger. Air waste causes hyperpnea and acapnia if the patient attempts to talk loudly and continuously.

Diagnosis.—The reflected and direct appearances are diagnostic of complete laryngeal paralysis. The diagnosis as to character and location of the causative lesion is made by following the twelve steps listed under "Diagnosis" in "Bilateral Midline Abductor Paralysis."

Treatment.—No treatment of any kind will have any effect whatever on the laryngeal paralysis. The patient can be made less uncomfortable by weekly aspiration of secretions from the tracheobronchial tree (q.v.) if they accumulate, as they usually do. The terminal phase, if the patient does not die of intercurrent disease or complications, is usually a drowning of the patient in his own secretions, because of inability to expel tracheobronchial accumulations. Bronchoscopic aspiration, followed by frequent catheter aspiration, will prolong life somewhat in such cases; but the prognosis as to duration of life is generally unfavorable.

Complications.—Pulmonary complications are the rule rather than the exception. The laryngeal paralysis itself may be regarded as a complication of the basic disease. Circulatory and metabolic complications are common. If the paralysis has followed thyroidectomy the possibility of endocrine disturbances (especially myxedema), tetany, and mental symptoms must be kept in mind.

Prognosis.—The prognosis as to improvement of function is hopeless, and the patient's life is likely to be shortened by pulmonary complications. Of course, the prognosis as to life may depend entirely upon that of the causative lesion, but neither return of motility nor change of cordal position ever occurs after a complete laryngeal paralysis, properly so-called.

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UNILATERAL MIDLINE PARALYSIS OF THE LARYNX

Loss of power of movement of one cord from the midline position due to impaired innervation is called "unilateral paralysis of the larynx." A common and convenient synonym is *posticus paralysis*. The term *abductor paralysis* is not incorrect but includes other than midline positions of the affected cord.

Unilateral midline paralysis is not an uncommon ailment though it is often overlooked. It is much more frequent than bilateral midline paralysis, and in our experience it is the most

INSPIRATION

PHONATION

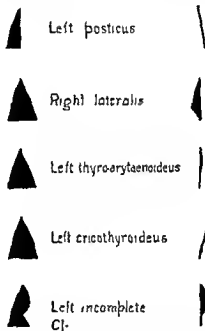


Fig. 394.—Glottic silhouettes in cases of unilateral laryngeal paralysis. Reflected images.

frequent of all forms of laryngeal palsy. The left side is affected approximately twice as often as the right.

Etiology and Pathology.—The causes are the same as those herein given for bilateral midline paralysis except that the unilateral type is much more frequently due to trauma than is the bilateral disability. It may be caused by an intracranial lesion affecting the laryngeal neurons between the nucleus ambiguus and the emergence of these neurons in the vagus through the jugular foramen (Fig. 334) or in the circuitous peripheral course of the inferior laryngeal nerve. The unquestionable clinical fact of greater frequency of left-sided laryngeal

paralysis is partly explained by the longer course of the left inferior laryngeal nerve and its deeper dipping into the frequently diseased upper thoracic region. Greater pathologic left sided vulnerability is applicable here only by theoretic implication. The mechanical reason for the midline position is that only the posterior crico-arytenoid muscle is affected. The edge of the cord is held firm and straight by the action of the unaffected cricothyroid, the lateral crico-arytenoid and thyro-arytenoid muscles.

Reflected Appearances (Fig 395)—Soon after the onset of the paralysis the midline position of one cord is conspicuous but after compensatory adjustment has taken place over looking of the disability quite commonly oc-

proximated cords may easily mislead the inexperienced examiner into finding nothing amiss. Fixing attention next solely on the glottis (*seriatim*) will however, reveal that the slit is slightly askew usually about 5 to 10 degrees from the sagittal line. When the turn comes to examine each arytenoidal mound it will be noted that on the sound side there is the normal bell crank movement whereas on the paralyzed side the mound droops limply forward. Thus drooping seems so much like an enlargement that the less experienced sometimes make a diagnosis of a tumor or an infiltration.

Appearances on Direct Laryngoscopy—The snapping shut of the glottis on phonation and cough by the action of one cord only with more or less of dragging of or pushing against

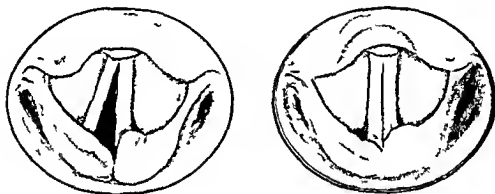


Fig 395—Reflected image seen in unilateral midline laryngeal paralysis. On inspiration the right cord moves outward normally while the left cord remains in the midline. The arytenoid eminence on the paralyzed side is tumbled over forward giving a tumor like appearance on inspiration but it is jostled back into place on phonation by the impact of the active arytenoid. Phonation is good in such cases.

curs. The only way to avoid this regrettable failure is to cultivate the habit of *seriatim* examination as advocated herein (see p 429). Thus examined it is readily noted that the unaffected side is the prime mover and that any movement on the paralyzed side is a secondary movement due to dragging or pushing by the overactive sound side, whose arytenoid carries its cord not only to but across the midline. In doing so it produces what we have found as one of the best means of diagnosis of unilateral paralysis namely *jostling of the arytenoid* on the sound side (Fig 395).¹ Watching each cord separately, by our *seriatim* method it is easily seen that the sound cord swings back more widely than is normal whereas the paralyzed cord moves only from the jostled position to the midline. On phonation, *E-e-e-e*, the ap-

pearances on the sound side and the slightly askew glottic slit on phonation are readily noted by the *seriatim* and checking-off system. The *mobility test* is next in order to determine the range of mobility of the respective crico-arytenoid joints. It is well also to slide the anterior commissure laryngoscope into the hypopharynx to note whether or not the cricopharyngeus is acting normally. It takes but a moment to note the appearance of the *cricopharyngeal crescent* (Fig 346) and it should always be recorded for immediate and future diagnostic use (see Syndromes Associated with Laryngeal Paralysis).

Symptoms—It is important to remember there may be no symptoms whatever if a unilateral paralysis has been present long enough for the establishment of good compensatory ad-

justment Usually the transient hoarseness at the date of occurrence has been attributed to a laryngitis that did not exist Close questioning as to such an attack may fix the date of onset There is usually no dyspnea even on moderate exertion A slight respiratory wheezing may be audible if the examiner's ear be placed close to the patient's open mouth If motor nerves other than the laryngeal are affected there may be symptoms from them, such as difficulty of swallowing in pharyngeal paralysis, or regurgitation of liquids through the nose in palatal paralysis Compensatory laryngeal adjustment usually enables the patient to cough up sputum effectively

Diagnosis.—The diagnosis of the laryngeal disability is readily made by the reflected and direct appearances noted above, using the seriatim and check-off system The character and location of the causative lesion can be determined by following all the steps enumerated under "Bilateral Midline Abductor Paralysis" (q v)

Treatment.—In uncomplicated unilateral midline paralysis no laryngeal treatment is required, but the patient and practitioner must both have in mind the danger that would be precipitated by paralysis of the other cord This sequela might require a prompt tracheotomy to save life (see "Bilateral Midline Abductor Paralysis") The patient should not go anywhere remote from tracheotomic aid His health should be maintained at the maximum Though often seemingly trivial, unilateral paralysis must always be regarded as a warning of potentially serious disease Causative lesions should be treated as indicated by their character and location

Prognosis and Sequelae.—Unquestionably, perfect motility returns in some of the cases of unilateral midline paralysis, in what proportion no one can say because in so many cases the existence of such a paralysis is overlooked In some the midline position of the cord is followed by the incomplete position In other cases a bilateral paralysis of the midline, the incomplete or the complete type, may follow The prognosis of each of these is given under the respective headings The prognosis of the causative lesion depends upon its character and location Prognosis should always be guarded, lest the patient wander far from tracheotomic help

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UNILATERAL INCOMPLETE PARALYSIS

The term "unilateral incomplete paralysis" denotes paralysis of all the laryngeal muscles of one side without paralysis of the arytaenoides "Cadaveric" as often applied to this condition is confusing and in some cases directly misleading Moreover, it is inapplicable because sagging of one cord only does not occur in the cadaver

Unilateral incomplete paralysis is not uncommon The left cord is affected oftener than the right, approximately twice as often in our experience

Etiology and Pathology.—The pathologic changes associated with the causative conditions are the same as those given for incomplete bilateral paralysis All the muscles of abduction and "tension" are paralyzed and the adductors are affected except the arytaenoides, which receives innervation from the nerve on the sound side, hence cannot be unilaterally paralyzed There is accumulating evidence to indicate that the lesion in primary unilateral incomplete paralysis is located in the vagus above the origin of the superior as well as inferior laryngeal nerves A lesion may impair laryngeal neurons without involvement of the entire vagus and this must be the case in bilateral incomplete paralysis because a lesion causing entire bilateral vagal destruction would be of fatal character Among our cases of unilateral incomplete paralysis the lesion was traumatic in six In two of these cases it was due to gunshot wounds, one perforating, the other penetrating, in the latter the bullet lodged near the basilar process after destroying the left vagus In the other four cases the paralysis was due to blows respectively by a pugilist's fist, a pitched baseball, a flying fragment of exploded emery wheel, some part of automobile wreckage In two other cases it was due to a growth in the nasopharynx—a carcin-

noma and a chondroma. The generally accepted statement that unilateral laryngeal paralysis does not occur in central lesions does not mean that such paralysis cannot be caused by an intracranial lesion between the nucleus ambiguus and the jugular foramen. If such a lesion is small the paralysis might be overlooked because the larynx is not examined when there is

lesion above the nuclei ambiguus causes only bilateral and spastic laryngeal paralysis but the laryngeal lower neurons originating in the right and left nuclei ambiguus, respectively, are intracranial until they enter the jugular foramen in the respective vagi. These neurons are often involved by compression or otherwise in the lesions of the various nearby centers, and such

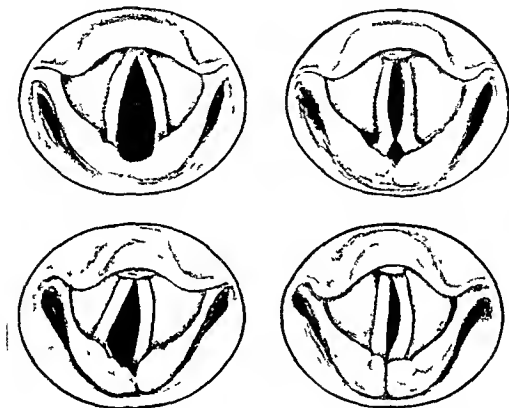


Fig. 396.—At the left above is shown the reflected appearance of a bilateral incomplete paralysis. The cords have concave edges and are motionless in a position midway between the midline and that of extreme abduction. This position is often wrongly called cadaveric and complete. The proper name for it is incomplete, because, obviously, there remains some power of movement in the arytenoides (cf Fig. 393). This form of paralysis may disappear, but usually it becomes a midline paralysis. The upper right hand drawing shows complete paralysis properly so called. It is never followed by recovery, nor by midline paralysis (cf Fig. 393). This appearance always means that the abductors, adductors, tensors and the tonus are all gone. The lower drawings show the reflected appearance of left incomplete unilateral paralysis. The right cord (left hand drawing) moves outward on inspiration leaving the paraboloid left cord motionless. Voice is poor with air waste because of paralytic defective approximation (right hand drawing).

no laryngeal dyspnea and little hoarseness as is so often the case in unilateral paralysis. In large intracranial lesions the gravity of the general condition of the patient is so extreme that there is no thought of laryngeal examination. In our experience an intracranial lesion has been the cause of unilateral laryngeal paralysis in the proportion of one case to eight cases of a peripherally located lesion. It is true that a central

involvement may be unilateral. These lesions being infranuclear are always nominally flaccid though, as previously mentioned, the pull of unparalyzed interrelated muscles may increase or decrease the apparent flaccidity.

Reflected Appearances.—During the first few months after the onset of unilateral incomplete paralysis (Fig. 396) the characteristic appearances are (1) the concave edge of the af-

fect cord, (2) its remaining stationary midway between phonation and approximation, (3) the paraboloid silhouette of the glottis on inspiration (Fig 394), (4) the crossing of the midline by the sound cord to meet the disabled cord, (5) the jostling of the sound arytenoid when struck by the active arytenoid in phonative or tussive approximation, (6) the upward passive displacement of the edge of the flaccid paralyzed cord by the expiratory blast

In the course of a few months these appearances will change by the adjustment that comes from the action of the unparalyzed muscles which are forced into excessive action by voluntary efforts of the patient to improve his husky voice. So great is this improvement that the paralysis is often overlooked in a casual examination in which the *seriatum* plan is not followed. Still further confusion results in a few cases from anomalous variations in the distribution of innervation, not only of the inferior laryngeal nerve but in the motor branch of the superior laryngeal as well. In some cases the full relaxation of the *arytaenoides* allows the vocal process to project, giving a *false impression of a unilateral complete paralysis*, a condition that is clinically nonexistent, because the *arytaenoides* cannot be unilaterally paralyzed (Fig 396). To determine the existence and degree of obliquity it is necessary to check on the plane of the mirror to see that it is not at a distorting angle.

Appearances on Direct Laryngoscopy.—One direct laryngoscopic examination (*q v*) should be made without locally-applied anesthesia to determine presence or absence of sensory paralysis and to note reflexes. Another examination should be made under local anesthesia (if needed) to study, *seriatim*, the anatomic parts and the movements. It will be thus seen that the paralyzed cord has no active movement, its apparent motion being a passive drag or push by the sound muscles. Soon after onset of the paralysis the glottis on cough and on attempted phonation has an approximately segmental silhouette. The curved vocal cord is the paralyzed one. After adjustment is established the glottic chink is narrow and its axis is more or less oblique. To determine the presence and degree of obliquity it is necessary to check on the long axis of the instrumental lumen to be sure it is vertical (patient recumbent). By the direct examination it is easy to determine the relative level of the cords, usually the disabled cord is

the lower. Flaccidity or spasticity are determined by the passive mobility test. This form of paralysis is practically always flaccid.

Symptoms.—The voice is husky until adjustment is established. This may require a few months or longer in some cases, but usually the voice becomes good, not quite as good as in midline paralysis, but better than in the bilateral incomplete condition. There may be a wheeze on respiration but there is usually no dyspnea as long as the other cord remains unaffected. In cases in which other paralyses are present their symptoms will be added to the clinical features. Pharyngeal and palatal palsies are the most frequent of these (see "Syndromes Associated with Laryngeal Paralysis").

Diagnosis.—Soon after its onset *seriatim* examination with the mirror will quickly reveal the laryngeal disability, as has just been described. After adjustment has taken place great care is necessary. Partial readjustment may mislead to a diagnosis of thyro-arytenoid paralysis (Fig 397). The distinctive feature is that in the latter condition the edges of the cord are relaxed and more flaccid, there is less width of chink than in the incomplete paralysis, even after adjustment. The steps in diagnosis of the causal lesion are the same as for bilateral midline paralysis.

Complications.—The most frequent and serious complication is paralysis of the sound side, making the paralysis bilateral. Unilateral incomplete paralysis is often only a part of a complex group of paralyses (see "Syndromes Associated with Laryngeal Paralysis"). Pulmonary complications are not likely unless the other cord becomes paralyzed.

Treatment.—It is of utmost importance to find and treat the causative lesion, not only because of the hope of benefit to the paralyzed cord but to prevent a bilateral paralysis by involvement of the other cord. Maintenance of the best possible state of health is important.

Prognosis.—In many cases perfect recovery occurs spontaneously. In many other cases the paralyzed cord assumes the position of a unilateral midline paralysis. In still other cases there is a satisfactory degree of adjustment for phonation and the other eight laryngeal functions that remains throughout life. The prognosis as to life is relatively good unless unfavorably affected by the causative lesion. Tussive pulmonary drainage is not a complicating problem as in the bilateral condition because glottic

cooperation in the tussive cycle soon becomes good by adjustment. The danger of asphyxia from the paralysis of the other cord is not great unless both cords should revert to a midline position and to the same level.

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THYRO-ARYTENOID PARALYSIS

Thyro arytenoid paralysis is defined as loss of motility of the thyro arytenoid muscle due to lack of motor impulses. This muscle is often called the internal tensor, hence the loose term "tensor paralysis," which covers other conditions also.

The *cause* is a lesion affecting the transmission of impulses through the motor neurons supplying the thyro arytenoid muscle. These neurons are bundled along with the other laryngeal neurons originating in the nucleus ambiguus of the same side (Fig. 334). Above that point the lesion would have to be large enough to affect both sides, and the result would be a bilateral spastic paralysis affecting also the external tensors, the cricothyroides, since supranuclear lesions affect movements, not individual muscles. Causative lesions are listed under 'Bilateral Midline Abductor Paralysis'. In the late stages of myasthenia laryngis there may be a degeneration of nerve endings in the muscles that may amount to a peripheral paralysis of the thyro arytenoides. These are the hardest worked laryngeal muscles in professional voice users and incessant talkers.

When both the thyro arytenoides are paralyzed, all other muscles being normally active, the *appearances in the mirror* are as shown in Figure 397. On inspiration both cords move

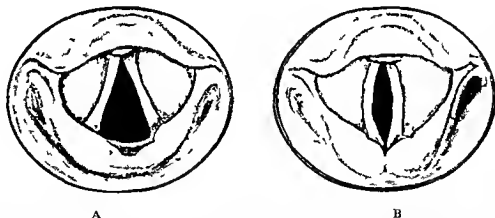


Fig. 397.—Thyro arytenoid paralysis. The reflected appearance on inspiration (A) is normal. On attempted phonation the paralytic condition of the thyro-arytenoides is shown by the bisegmental glottic silhouette (B). It was further evidenced at the examination of this patient by the wafting up of the unhardened edges on phonation. The voice was weak and husky, with a rasping sound of air waste.

As an independent condition thyro arytenoid paralysis is not common as part of a complete paralysis (*q 1*) it is not infrequent in our experience. In most of the cases reported the condition was evidently a fatigue of the overworked thyro arytenoid muscles part of a myasthenia laryngis (*q 1*) not a paralysis. In some reported cases the illustrations show the real condition was, obviously, an incomplete paralysis, that is, the abductors were also paralyzed in addition to the adductors, except the arytenoides

outward to the normal inspiratory position the glottic silhouette is a triangle. On attempted phonation the cartilaginous glottis is closed by the pull of the arytenoides, the membranous glottis is nearly closed by the laterals but a narrow slit is left by the symmetric curve of the cordal edges. The glottic silhouette, when one cord is affected, is a segment of a circle of long radius (Fig. 394), in the bilateral condition the silhouette is made up of two such segments with chords (geometric) together (Fig. 397). On

saying "E-e e-e" the sound is weak and low in pitch for the individual under examination

The view through the *anterior commissure laryngoscope* is vivid. From the wide *inspirational* position the arytenoid cartilages snap together normally but the cordal edges lag and obviously are unable to firm as do normal cords.

In addition to a hoarse quality the voice has the significant *symptom* of low pitch. There may be accompanying symptoms of associated paralysis (see "Syndromes Associated with Laryngeal Paralysis")

The appearances just mentioned are *diagnostic*. Due allowance for individual variation in the firming of the cordal edges must be made. In some individuals with good ordinary voices, approximation is not as close as in others. In *myasthenia laryngis* the cords are firmed for a moment and then they relax as the voice breaks or pitch drops. In *tuberculosis* the anterior commissure laryngoscope will detect an infiltration in the posterior commissure. The presence of a subglottic tumor producing a similar appearance, and invisible in the mirror, is likewise revealed.

Apart from the causative lesions the disability of other laryngeal muscles, especially the postici, the arytaenoides, and the cricothyroides, is the most common *complication*. The thyro arytaenoides are included in a complete paralysis.

Treatment is directed primarily to the causative lesion. To promote readjustment of laryngeal structures and to retard atrophy, use of the voice is essential. Overuse and fatigue must be avoided. Constant supervision is required to prevent development of *myasthenia* in the unparalyzed muscles. There should be at least two hours of absolute silence during the day to supplement the usual eight hours of rest at night (see "Prophylaxis" under "Myasthenia Laryngis"). Violent forcing of the voice as in attempted shouting should be prohibited. Heat treatment by high wattage lamp or reflected sunlight over the larynx externally is helpful.

In the *prognosis* the causative lesion is the most important element to consider. In many, perhaps in half of the thyro arytenoid paralyses, recovery takes place if the cause is removed early. Readjustment restores fairly good voice, as well as other laryngeal functions, in permanent cases. Paralysis of the postici or of all laryngeal muscles (see "Complete Paralysis of the Larynx") may follow a thyro arytenoid

paralysis, as one of the exceptions to Semon's rule.

Unilateral Thyro-arytenoid Paralysis—The thyro arytenoid muscle of one side is sometimes encountered as an isolated affection, but in most cases it is bilateral or is part of a complete or incomplete paralysis, as mentioned in consideration of these subjects. The causes, diagnosis, and treatment are the same as given for the bilateral condition, which is sometimes a sequela. The voice is not so husky and adjustment comes sooner and is better in the unilateral condition.

CHEVALIER JACKSON

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SYNDROMES ASSOCIATED WITH LARYNGEAL PARALYSIS

A syndrome is a complex of symptoms. In laryngologic literature it has been customary to apply personal names (not always justly) to groups of paralyses when laryngeal paralysis forms a part. The result is much confusion, when progress of the disease transfers it from one man's syndrome to another, and in the end no name will fit. Yet the word "syndrome" is very useful for brevity and as a reminder to be on the alert for associated paralyses. When they fit they are very convenient, when they do not the numbers of the cranial nerves, or the region in which the causative lesion is located, should be used.

Syndromes associated with laryngeal paralysis are not uncommon.

Etiology and Pathology.—Mechanically speaking the cause is associated involvement of neurons other than laryngeal. The most frequent site of the causative lesion is in the nucleus ambiguus or somewhere between that point and another a few centimeters below the jugular foramen. In this region the last four cranial nerves, all bulbar in origin, are close enough together for a relatively small lesion to affect two or more of the group (Fig. 398).

The glossopharyngeal (ninth) supplies the superior constrictors and palatal muscles and taste to the tongue posteriorly. The pneumogastric (tenth) supplies sensation to the laryngeal mucosa and motion to cricothyroid and arytenoid muscles and sensation to the palate and pharynx through the superior laryngeal also motion to all other muscles of the larynx through the inferior laryngeal. It is also a cardiac accelerator. The spinal part of the spinal

aortic arch. The most common groupings are associated with the names listed in Figure 398. Regardless of the names the drawing will simplify the study of any combination of paralyses.

Designations—*Syndrome of Collet Sicard*—This is also called *Villaret's syndrome* and designated involvement of the cervical sympathetic as well as the last four cranial nerves producing paralysis of larynx, tongue and soft palate in

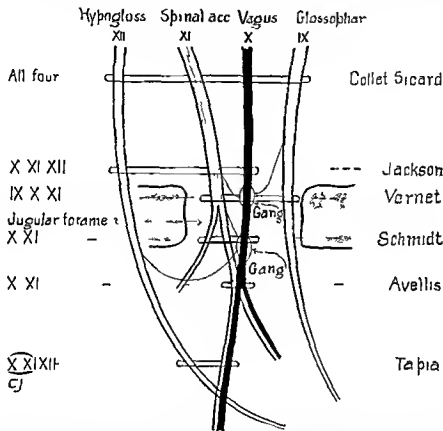


Fig. 398—Simplified schema illustrating the anatomic factor in the syndrome of the jugular foramen and other syndromes associated with laryngeal paralysis. The vagus (Xth) encloses the fibers of all the laryngeal nerves in close association with the spinal accessory, glossopharyngeal and hypoglossal nerves. The loops around groups represent lesions involving two or more of these four cranial nerves, not necessarily at the precise location shown. The lesions may be nuclear. The names at the right are those usually attached to the respective syndromes. Apart from names, this schema taken with the function of the respective nerves will simplify the study of any complex case of paralysis by a lesion in this region.

accessory (eleventh) supplies the sternomastoid and trapezius muscles; the accessory part, the laryngeal muscles and palate. The spinal accessory supplies motor roots to the vagus; the two vagal ganglia supply sensory roots. The hypoglossal (twelfth) is purely motor; it supplies all the muscles of the tongue except the palatoglossus. Important anatomic regions traversed by the nerves involved in syndromes are the bulb, jugular foramen, parapharyngeal space

the sphere of the laryngologist in addition to the trapezius and sternocleidomastoid (Fig. 398).

Syndrome of Hughlings Jackson—In this syndrome, in addition to the laryngeal paralysis, there is paralysis of a half of the soft palate and tongue, also of the sternocleidomastoid and trapezius muscles, all on the same side (Fig. 398).

Syndrome of Vernet—In this syndrome there

is paralysis of the larynx, pharynx, soft palate, and sternocleidomastoid and trapezius muscles. Accompanying sensory paralyses are anesthesia of the larynx, pharynx, soft palate, and loss of taste in posterior third of the tongue. The pharyngeal paralysis causes difficulty in swallowing (Fig 398).

Syndrome of Schmidt—Paralysis of the larynx along with paralysis of the sternocleidomastoid and trapezius muscles, all on the same side, is known as Schmidt's syndrome (Fig 398).

Syndrome of Avellis—In this syndrome there is paralysis of the larynx and palate on the same side. Usually there is more or less trouble in swallowing due to involvement of pharyngeal constrictors. There may be loss of pain and temperature sense on the opposite side including the extremities, trunk, and neck. When this syndrome is complicated by eye symptoms—sinking in of the eyeball, ptosis of the upper lid, slight elevation of the lower lid, and contraction of the pupil—it is known as the *syndrome of Avellis-Horner*. The name of Avellis was applied to softening of one side of the medulla due to thrombosis of the anterior spinal artery, a branch of the vertebral, but it is now used for any lesion involving the nuclear or lower neurons indicated in Figure 398 (see also under "Paralyses in Relation to the Esophagus").

Syndrome of Tapia—In this syndrome the larynx and tongue are paralyzed on the same side, if the palate is also affected it is on the same side. This syndrome is more frequently due to tumor or wound of the neck than are the other syndromes (Fig 398).

Reflected and Direct Appearances.—In the larynx the appearance may be any of those described in connection with the various forms of laryngeal paralysis on preceding pages. The anterior commissure laryngoscope, after laryngeal examination is complete, should be passed into the hypopharynx in order that the cricopharyngeal crescent may be examined (Fig 346). The crescent is flat or absent when the cricopharyngeus is paralyzed, and usually the inferior constrictor is also paralyzed.

Symptoms.—The laryngeal symptoms have been fully covered in dealing with the respective laryngeal paralyses. The symptoms other than laryngeal are indicated in the description of the respective syndromes just given.

Diagnosis.—The diagnostic methods to be applied in the study of syndromes are precisely those listed under the heading of "Bilateral Midline Abductor Paralysis", and it might be added if that list were always categorically followed syndromes would not be overlooked as they sometimes are in their minor phases. For emphasis it may be repeated here that the action of the pharyngeal muscles, especially the cricopharyngeus, should always be noted at the direct laryngoscopic examination. It takes but a moment to slide the instrument into the hypopharynx when a slight forward lift will expose the cricopharyngeal crescent on the posterior wall (3, Fig 346). The passive mobility test with the anterior commissure laryngoscope is invaluable for diagnosis in these cases, and at the same time a paralysis of the constrictors and of the cricopharyngeus can be determined.

Treatment.—The causative lesion usually gives the most important indication for treatment. This and the treatment of the laryngeal paralysis has been covered in preceding pages. When involvement of the pharyngeal muscles interferes with swallowing the feeding tube passes readily. It should be in the form of a catheter passed through the nose. A carefully-planned full diet in liquid form must be insisted upon and strictly followed on written schedule, otherwise these patients will suffer from inanition and avitaminosis. An excess of mixed vitamins continuously maintained should be added to whatever medication is indicated. These precautions as to diet and vitamins alone are sufficient for diphtheritic cases, it is always imperative that they be added to specific treatment in syphilitic cases, and to postoperative treatment in surgical cases.

Prognosis.—The causative lesion and the duration of the paralysis are the two factors that bear upon prognosis. Other things being equal the chances of the recovery of laryngeal motility are inversely as the duration of the disability, regardless of the cause.

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ABDUCTOR PARALYSIS IN THE NEWBORN

Abductor paralysis in the newborn is congenital paralysis of the posticus on one or both sides. *Congenital laryngeal stridor* and *blue baby* are synonyms applicable to some cases.^{1, 2} The disability is not uncommon but is usually overlooked. The return of motility in almost all infants who survive indicates that the cause was either bulbar circulatory disturbance or pressure on nerve trunks. Delivery was with forceps in almost all cases we have seen, this clinical fact points to trauma as the chief cause.¹ A few of the patients were syphilitic. The reflected appearances are unknown because the use of the mirror is impracticable in infants. The appearances on direct laryngoscopy are described under "Asphyxia Neonatorum." The symptoms of cyanosis, dyspnea, and stridor are intense in the cases of bilateral paralysis. Stridor and a hoarse cry are the chief symptoms in the unilateral cases. The only means of diagnosis is examination with the direct laryngoscope. Failure to abduct is obvious. The cords remain in either the midline or the incomplete position. If asphyxia is impending, as it always is in bilateral midline cases, a bronchoscope should be inserted, to get the breathing well established and to obtain good color. Insufflation through the bronchoscope, of oxygen (with 5 or 7 per cent carbon dioxide admixture) will help greatly in this. A very gentle flow of the gas is essential for safety. Then the diagnosis can be confirmed by the snapping shut of the cords as the bronchoscope is withdrawn. The passive mobility test (*q v*) will then complete the diagnosis. Presence or absence of congenital syphilis should always be determined. A reasonable degree of care in the use of obstetric forceps would probably eliminate trauma to the laryngeal neurons, except in such emergency cases as require prompt delivery. The treatment consists in prevention of asphyxia. When it is obvious that the laryngeal obstruction would cause asphyxia, low tracheotomy should be done with the bronchoscope in situ. The technic is given under "Tracheotomy." Humidity of the air of the room should be maintained at or above 65 per cent, by a mechanical humidifier, or an electrical water heater, or a boiling water kettle. Occasional catheter aspiration (*q i*) by the nurse will probably be advisable. Congenital syphilis, if present should be treated (see p 21). Practically

all patients recover perfect motility and laryngeal function. No difficulty in decannulation is encountered when the tracheotomy (if this operation has been required) is done low, as it should be. The only danger to life is that of asphyxia.

CHEVALIER JACKSON

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- 2 Jackson, Chevalier. *Peroral Endoscopy and Laryngeal Surgery*. St. Louis: The Laryngoscope Company, 1915.

TRAUMA OF THE LARYNX

Trauma of the larynx includes wounds or injuries of the larynx other than surgical. Operative wounds, being aseptic, are not in the same class. A laryngeal injury may be called direct when inflicted by an external agent, or indirect when due to action of the laryngeal muscles. The latter class has been considered under the headings "Myasthenia Laryngis" and "Contact Ulcer of the Larynx." *Traumatic laryngitis* is synonymous but not distinctive.

In no part of the literature of traumatology is there such a dearth of data as in wounds of the larynx. Most of the cases are buried in the broad classification of injuries of the neck. Mortality statistics afford no data whatever on deaths from asphyxia or hemic drowning in cases of cervical trauma involving the larynx under conditions in which immediate surgical attention is impossible.

Etiology.—The larynx is protected posteriorly by the cervical part of the spinal column and above by the skull, in front it is partially protected by the projection of the chin and the anterior wall of the thorax. Still more is it protected by its resilient suspension. It is widely exposed on both sides, and in front it is exposed to being injured by any object of such shape or position that it can get between the chin and the thorax. As a matter of fact, the protection of the larynx and its escape from injury are both exaggerated in an effort to explain partly in the records which, as stated, is really due to the fact that nearly all cases of laryngeal injury are recorded under the general classification of in-

juries of the neck. Available records of personal experience of fifty odd years show that the causes of laryngeal trauma have been classifiable as follows:

1 Highway accidents (a) gash by shattered wind shield, (b) contusion by striking of neck on rim of steering wheel in collision, (c) fracture of thyroid cartilage by striking of neck on unknown part of wreckage.

2 Railway accidents. Train crew (a) catching of neck on a sagged wire, (b) crushing of neck in hand coupling of cars, (c) striking of neck by handle bar of handcar. Passenger (d) striking of neck on back of forward seat in collision.

3 Industrial accidents (a) burn by live electric wire, (b) premature blast in quarry, (c) fall on moving gears while intoxicated, (d) striking of neck on end of pick handle when stooping in dark, (e) burns from inhalation of chemical fumes and hot smoke, (f) burns from swallowed battery acid, probably dilute sulfuric.

4 Household accidents (a) fall with neck striking edge of step, table, chair, stepladder, (b) burns from swallowed caustic agent (lye).

5 Farm accidents (a) kick of a horse, (b) trampling by horse's hoof.

6 Medical and surgical accidents (a) stabbing tracheotomy, (b) misdirected intubation tube, (c) aggressive endolaryngeal surgery, (d) radium and roentgen ray overdosage, (e) forceps delivery.

7 Sporting accidents (a) trampling by football player, (b) impact of golf ball, (c) impact of baseball, (d) striking neck on sled while coasting.

8 Brawls (a) blow of fist, (b) gash with backed razor, (c) stab with ice pick, (d) gash with cleaver, (e) gash with butcher knife.

9 Police encounters (a) perforation by bullet, (b) gash with knife, (c) blow with lead pipe.

10 Suicidal attempts (a) crushing and displacing compression by hanging, (b) gash with razor, (c) forcing of two foreign bodies, a key and a screwdriver into the larynx.

Pathology.—Occlusion of the lumen of the larynx may occur immediately without inflammation. The fundamental anatomic fact is that the lumen of the larynx is normally not much larger than is required for respiration. Therefore relatively little encroachment on the lumen is a serious matter and a great mass is not required to produce asphyxia. Sufficient tissue to occlude the lumen may be displaced at the moment of injury, resulting in immediate asphyxia. Occlusion may come on in a few minutes from submucosal hemorrhage, a hematoma. An hour or more later the lumen may be occluded by submucosal emphysema. This may be accompanied by mediastinal emphysema, aided by muscular pressure and thoracic suction, the air quickly finds its way downward between the layers of cervical tissue. Subcutaneous emphysema may or may not co-

exist. Simultaneous wounding of adjacent large vessels may produce a flow of blood large enough to enter the trachea and drown the patient. The foregoing processes are *noninflammatory* and must be borne in mind as immediate emergencies. Inflammatory reaction developing later may cause occlusion of the laryngeal lumen by the well known "edema of the glottic tissues." As with traumatic lesions elsewhere, a wound of the larynx may be in the form of a crush, a contusion, an incision, a laceration, a puncture, a penetration, a perforation, a fracture, or a dislocation. The traumatizing agent may remain, may have passed on, or may have been withdrawn, in either of the latter two happenings it may or may not have left extraneous matter. There may be associated injury of adjacent tissues, or the larynx may be severely injured with no evidence of any other trauma nearer than the shoulder. Traumatic laryngectomy with survival has been reported. In a case in our experience the entire larynx of a quarryman was torn away by a premature blast. Doubtless there are many fatal cases in which the larynx has been destroyed along with a part of the neck. There are three classes of cases peculiar to the larynx, namely, (1) those in which the cartilage and its perichondrium are uninjured, (2) those in which these structures are damaged, and (3) those in which there is involvement of the hypopharynx or of the cervical part of the esophagus. Other phases being equal, prognosis and treatment in any given case are dependent on the *determination of which one of these three classes covers the particular case*. The pathologic course, clinical features, and duration in each class are quite different. If the cartilage and its perichondrium have not been injured, recovery is usually prompt, and complications, if any, are only those of any wound. If, on the other hand, the cartilage and its perichondrium are involved, there is almost certain to be perichondritis, prolonged suppuration, chondral necrosis, and building of cicatricial tissue in which subsequent contraction unresisted by a cartilaginous framework ends in chronic laryngeal stenosis, even total atresia. Reparative processes of cartilage anywhere are poor, poorer even than those of bone. In laryngeal cartilages of adults union is fibrous and rarely, if ever, cartilaginous. In the third class of cases, those in which there is an opening from the wound into the hypopharynx or the cervical part of

the esophagus, the wound is deluged with oral secretions, food, and fluids, containing all the infective agents of the mouth, tonsils, nasal passages, and accessory cavities. If there have been extensive lacerations, the wound may be covered with grayish sloughs similar to those usually formed after tonsillectomy. Unless controlled by antiseptics, such a wound is a foul mess. *Fracture*, if due to garroting or to a blow, usually involves the partially ossified anterior part of the thyroid cartilage where the wings meet, the superior horns may be broken off. This form of injury is often accompanied by fracture of the hyoid bone, the cricoid cartilage, which is a complete firm ring, is more resistant. Missiles, penetrating or perforating, may shatter any of these structures, or may only plough a groove in the external surface. In a case in our experience, that of a policeman, both arytenoid cartilages were shot away by a perforating bullet. Any injury to laryngeal cartilage may or may not be followed by emphysema but is almost invariably followed by perichondritis, chondral necrosis, prolonged suppuration, and, if not prevented, cicatricial stenosis or even total atresia. *Injury to the inferior laryngeal nerve* in trauma of the neck, causing abductor paralysis without direct injury to the larynx itself, is rare, but we have seen two cases. In one the injury was unilateral, in the other, bilateral. The superior laryngeal was injured unilaterally in three other cases causing typical paralysis of motion but not of sensation. *Dislocation* occurs from an extremely forcible displacement of the cricoid cartilage backward alongside the vertebral body. *Rupture of the ligamentous capsule of the cricoarytenoid joint* probably occurs in most cases. In a case which we encountered, both arytenoid cartilages were dislocated backward in a fall against the top rail of a fence. In another case a similar double dislocation was caused by explosion of an autotruck tire.² In a third patient, who had attempted to commit suicide by hanging the cricothyroid and both cricoarytenoid joints were dislocated by the jerk of the rope. *Burns* of the larynx may complicate deep burns of the neck, endolaryngeal burns may be produced by inhaled flame, hot smoke, or caustic vapors. We have seen many cases in firemen.

Symptoms—Any one or more of the following symptoms may be present in a case of injury involving the larynx: hemoptysis, blood-

spattering cough, hematemesis, nausea, hoarseness or aphonia, dyspnea, stridorous, hissing, wheezing, bubbling, gurgling, or rattling respiration. Local pain is usually moderate. There may be cyanosis, more often there is ashy gray pallor even if there is obstructive dyspnea. Locally the skin of the neck immediately after injury may appear normal. There may be ecchymosis, swelling, tenderness, hematoma, or subcutaneous emphysema. If the larynx is obstructed, there will be indrawing at the suprasternal notch. If there is an open wound of the neck, the larynx and trachea may be laid bare in the wound. If either has been opened, the blood will be bubbling, and it will be spattered by coughing. An opening into the pharynx reveals itself by a flow of thick glairy saliva, and when water is taken the wound is flooded, if there is any attempt to swallow food, it is projected out over the wound. The general symptoms are the same as in injury elsewhere. The patient may be unconscious from loss of blood, from shock, or from concurrent injury of the central nervous system. There is no special laryngeal shock, as was once thought and taught, in connection with surgical operations.

Diagnosis—The symptoms may or may not be suggestive, but examination of the larynx is indicated in every case of injury to the head or the neck, even in the absence of any laryngeal symptom. All four methods of examination—by mirror, by direct laryngoscope, by palpating finger externally, and by roentgen ray—are necessary for completeness. There may be circumstances under which all four of these methods cannot be used immediately, the attendant must then do the best he can, remembering always that the laryngeal examination is incomplete and that thorough examination by all four methods must be done as soon as circumstances permit. The diagnosis of obstructive laryngeal dyspnea can always be made objectively by the indrawing at the suprasternal notch, at the clavicles and in the intercostal spaces. When these signs are present in any case of trauma in or near the larynx, low tracheotomy should be done without waiting for completion of the delayed diagnosis. One diagnostic method, palpation, is always promptly available, and it is invaluable. When the skin is not destroyed and before swelling has obliterated landmarks, the thyroid and cricoid cartilages can be palpated in their entirety by trained

fingers, and in traumatic conditions, as well as in disease, information otherwise unobtainable can be elicited with certainty. A *fracture* may be felt as two edges where there should be continuity. This usually means that the perichondrium has been ruptured, but not always. Crepitation may be elicited as a sensation, rarely as a sound. It is readily distinguished from the crackling sensation of emphysema by its giving a sensation of displacement of the air bubbles under digital pressure. Emphysema and fracture may coexist. In case of multiple fracture producing fragmentation the flexibility where there should be relative rigidity is unmistakable to the trained touch. The roentgen ray (*qv*) is an invaluable diagnostic means and should be used as soon as possible in every case of laryngeal injury, obvious or suspected. When there is an open wound of the neck involving the larynx, complete exploration is best deferred until it can be done under aseptic precautions except so far as the control of hemorrhage or the forestalling of asphyxia may necessitate. Reflected appearances will depend on the character of the injury and the time elapsed. A crush may show a crowding of the tissues into the airway. Soon there will be mucosal ecchymosis or submucosal hematoma, giving a reddish, purplish, or violet color, sometimes this is accompanied by blood or blood tinged secretions. Soon, also, there may be submucosal emphysema with pallor of the mucosa. Some conditions, such as displaced tissues, blood, hematoma, emphysema, may cause *asphyxia before the onset of inflammation*. The appearances of traumatic laryngitis may be those of reddish mucosal inflammation or whitish watery edema, or there may be a visible cut or an obvious laceration. If the trauma is entirely submucosal, there will be, after a few days, supuration with development of a paler color at the site of impending breaking through of pus. A *gash* from a sharp-cutting edge, such as we have seen a number of times from a backed razor and once from a cleaver, also from a butcher knife, at first is easily overlooked. Soon, however, the cut edges swell, evert, and are covered with exuberant granulations, sometimes with exudate. A *penetrating spent bullet* may be visible endolaryngeally as a bluish spot under the mucosa. We have seen a perforating bullet track with an endolaryngeal wound of entrance on one side and one of exit on the other. These openings were associated with a

track from one side of the neck to the other. In case of *stab* with an ice pick the appearances were similar. There was a wound on each side of the neck, over the central portion of the left ala of the thyroid cartilage and at the posterior edge of the right wing. The endolaryngeal wound on the left side was in the ventricle, that on the right side was anterior to the arytenoid cartilage. It seemed probable that the ice pick had entered on the left side of the neck and emerged on the right, but this could not be positively determined. Palpation revealed a *comminuted fracture* of the thyroid cartilage.

Complications.—It has become a tradition to enumerate pneumonia as a complication of trauma, even surgical trauma, of the larynx. Bronchology has taught us, however, that in many instances the so-called pneumonia is really atelectasis (without pneumothorax) due to bronchial obstruction caused by a thick, tenacious secretion of such high viscosity that the cilia and the cough reflex cannot expel it. Over and over again we have seen all the physical signs on which the diagnosis of pneumonia was based disappear after bronchoscopic aspiration. So often has this occurred that we feel skeptical of any diagnosis of pneumonia in such cases unless confirmed by diagnostic bronchoscopy. Precisely the same may be said of edema of the lungs. Mediastinal emphysema is a common complication. It may be the result of either of two different mechanisms seldom of both combined. With the skin of the neck broken, subcutaneous cervical emphysema may (more or less aided by muscular pressure and thoracic suction) extend between the tissue layers into the mediastinum, or with a large open wound involving the suprasternal notch, the inspiratory intrathoracic negative pressure may suck in air whose escape is prevented in a valvular way. That is to say, the air is drawn in on inspiration, but exit is hindered by the prompt crowding of the tissues into the thoracic aperture. The result is that intrathoracic pressure progressively increases until an atmospheric degree of pressure is reached and the patient is asphyxiated because no more air can be drawn in, or until the patient dies of positive pressure effects on the heart, the circulation, and the innervation.

Association with Other Injuries or Diseases.—Wounds of any portion of the body may, of course, accompany wounds of the larynx, but most frequent are wounds of the neck. Of ut-

most clinical importance are wounds involving the pharynx and cervical esophagus. Psychic laryngeal trauma (*qv*) may be present due to the same incident. Most important is systemic syphilis. As soon as the urgent indications have received proper attention a serologic test should be made because of the bearing of syphilis on repair of cartilage and the development of stenosis.

Treatment—Fundamental to all treatment is the anatomic and physiologic fact that the larynx is located on "the lines of communication" on which life of the patient depends, the air and food passages. The cardinal rule of traumatic surgery places arrest of hemorrhage as the first requirement. In trauma of the larynx the primary indication is double "Don't let the patient die of asphyxia or hemorrhage." The cardinal signs of obstructive laryngeal dyspnea (*qv*) call for immediate low tracheotomy. But *tracheotomy for hemorrhage* is also often an urgent indication under circumstances parallel to those calling for the emergency use of the tourniquet in traumatic hemorrhage of the extremities when search for bleeding vessels in the wound is not practicable. Obviously an ordinary tourniquet cannot be used on the neck. A potentially life saving substitute is firm compression with use of a properly placed tracheal cannula to maintain the airway. Of course, excessive pressure on both common carotids must be avoided. In the absence of indrawing, tracheotomy should always be borne in mind as a requirement that may be urgently indicated at any time. Apart from dyspnea it may be indicated for (1) hemorrhage, (2) cervical emphysema to forestall mediastinal emphysema, (3) mediastinal emphysema, (4) relief of tracheo-bronchial compression of any kind, (5) aspiration of inspired or flowing blood, and removal of clots or other foreign material, if peroral bronchoscopy is unavailable, (6) insufflation of oxygen (plus carbon dioxide, 7 per cent), (7) relief of pulmonary complications by frequent catheter aspiration, (8) thoracic penetration or perforation, in some cases.

Endolaryngeal hemorrhage is controlled, if severe, by a gauze packing of the larynx through the direct laryngoscope after a low tracheotomy for breathing.

Endolaryngeal wounds usually require no local application when they do not communicate with an external wound. If desired a little bismuth subnitrate may be insufflated. The

amount must be very small and free from lumps. The same is true of the sulfonamides.

Care of the External Wound—In the absence of hemorrhage or impending asphyxia, or in case these emergencies have been attended to, the wound must be given proper attention. In external wounds of the neck involving the larynx prevention of contamination is of utmost importance. This indication is best met by dusting the wound with one of the sulfonamides, a small pad of sterile gauze being momentarily held over the laryngeal or tracheal opening to prevent the entrance of an excessive amount of the chemical into the air passages, the same would apply to the food passages if they are also involved. The relative merits and dosage of toxic sulfonamides and of the nontoxic penicillin and other bacteriostatics locally and systemically useful are fully discussed under the head of "Chemotherapy in Otolaryngology." Ordinarily at the first dressing, to protect the wound from contamination, any available sulfonamide is used for a light dusting and a sterile gauze dressing is applied. In the subsequent care of the wound the outstanding indication is for the prevention of laryngeal stenosis. Debridement is contraindicated in the larynx. Ordinarily the wound cannot be cleaned and closed. It is better to pack the wound open until infection is controlled and necrotic cartilage, if any, has come away spontaneously.

Treatment of Crush, Fracture, and Dislocation—In a crush of the laryngeal box the cartilages may be bent, fractured, or fragmented. The whole future usefulness of that larynx depends then on prompt and proper care. The best form of treatment is that employing core molds, they serve as internal splints on which the natural form of a laryngeal lumen is maintained or rebuilt. As a rule, the sooner the core mold is placed the better will be the result. Early placement is especially imperative, because the deformity usually associated with a crush will soon become fixed, and many months may be required for restoration, whereas if a properly fitting core mold is put in place immediately, the structures are restored to their proper locations and become fixed there, thus establishing a good laryngeal framework. The core mold should not be as close a fit as is necessary to promote absorption in dealing with cicatricial conditions of long standing. Simple fracture of the laryngeal cartilage, without fragmentation

or crushing, and not compound, usually calls for immediate insertion of a core mold as an internal splint. It should not be of too large a size. If there is much endolaryngeal swelling, it should be a rather close but not tight fit in the narrowed lumen. Larger sizes can be progressively substituted as the swelling subsides. This subsidence will be rapid as compared with that of cicatricial tissue in chronic stenosis, unless the fracture is compound, in this case there is always perichondritis with suppuration ending in chondral necrosis. Here again core molds are the best form of treatment, as mentioned in another paragraph.

Dislocation of one or both arytenoid cartilages can be reduced by the insertion and wearing of a cord mold of large diameter if the patient is seen within a few days after the accident. For this, tracheotomy must be done, if it has not already been performed for prevention of asphyxia. If the dislocation has been unrecognized until fixation in the dislocated position has occurred, reduction with restoration of good movement is impossible. The selection of the proper time for beginning the core mold treatment is of utmost importance in trauma of the larynx. If there is a fracture of the thyroid or of the cricoid cartilage without any damage to the skin or the mucosa, a core mold of an easy-fitting size may be inserted at once. If, however, there is trauma of the interior of the larynx, the first core mold should not be inserted until the virulence of the infection has been determined or until the necrotic tissue has sloughed off and the raw surfaces are covered with healthy granulations. This, under treatment with a sulfonamide or penicillin, will ordinarily require only a week or two, unless the infection is exceptionally virulent. Epithelization should not be awaited before beginning the insertion of the core molds; these are the best means of promoting the essential epithelization with epidermal epithelium. If the patient does not come under observation until after a longer period has elapsed and the building up of inflammatory tissue has begun to narrow the laryngeal lumen, the core mold treatment should be begun at once. If the patient comes at a still later period, the case may be one of cicatricial stenosis or atresia (*q v*). The technic of insertion and withdrawal of core molds is given in connection with these subjects.

Involvement of the Pharynx and Cervical

Esophagus—When the trauma or subsequent sloughing has opened into the pharynx or cervical esophagus prompt and proper care is necessary to save life. Two measures are essential: (1) A soft rubber catheter of smaller diameter must be worn constantly through the nose, the distal end being in the thoracic portion of the esophagus. This tube serves four purposes: (a) it promotes esophageal drainage of secretions alongside it, (b) it enables maintenance of proper feeding and intake of fluids, (c) it prevents dumping of food into the wound, and (d) it prevents total atresia of the pharynx and of the cervical portion of the esophagus. The last objective is a prime necessity, because any lumen, however small, can be safely dilated, whereas total atresia requires a perforating operation locally as well as gastrostomy externally. (2) There must be maintenance of a fully balanced diet and of an adequate intake of fluids. The total of a perfect formula must be taken each day, otherwise resistance will be lowered and healing will be retarded. (See additional details under "Compression Stenosis of the Esophagus" and "Trauma of the Esophagus".)

Plastic Repair—One of the reasons for the words of caution regarding debridement is the necessity for tissue for plastic repair. If only devitalized tissue has been removed there may be material with which to work. For deficiency in the form of an open trough in the airway there must be no drawing together of the margins of the gap, and soft-tissue repair will collapse on inspiration. For stiffening it is necessary to transplant cartilage. The most satisfactory way is to implant cartilage of the desired size and shape in a selected adjacent area as a sterile procedure, after the cartilage has healed in, the pedunculated flap containing it is swung into place, forming a lid to the laryngeal trough which is thus propped open. In cases in which the pharynx or the cervical esophagus is involved repairs are made in the same way as in laryngectomy for cancer of the larynx (*q v*).

Treatment of Burns—Burns of the neck deep enough to affect the larynx are usually fatal. If not, the treatment consists in preventing contamination, or, oftener, in the control of infection. The sulfonamides are useful for this, they are used systemically, as well as locally in form of light dusting. The amount must be controlled and toxic concentrations must be

avoided as mentioned in the section on specific drugs. Endolaryngeal burns are treated differently. Searing or scorching by inhaled flames, hot smoke, or caustic vapors usually produces a thick, tenacious secretion from the laryngotracheobronchial mucosa. This secretion should be aspirated to prevent asphyxia. Apart from relieving bronchial obstruction, the clearing of the airway permits local medication. The best form of medication is a spray of a mild solution of sodium bicarbonate; it is not only soothing but also facilitates aspiration by loosening secretions and exudates. Both aspiration and spraying can be done best with the direct laryngoscope or the bronchoscope. The technic is shown in Figures 469, 470, and 471 (pp. 628, 629). If these are not available, tracheotomy should be done, even if not required for dyspnea. It has the added advantage that the aspiration and the medication can both be done by the nurse, as often as necessary, every half hour or oftener. For aspiration the nurse uses a soft rubber catheter through the outer cannula.

General Care—Transportation, the treatment of shock, transfusion, and general care and management are part of general medicine and surgery. One important point requires emphasis here, namely, in cases of disease or injury of the larynx or tracheobronchial tree all opiates and atropine are strongly contraindicated because they inhibit natural peroral drainage by cough, tussive squeeze, and ciliary action. By thickening the secretions they clog the cilia. Casualties should be promptly tagged "Don't give morphine or atropine." 2 3 4

Prognosis—In the early stage of laryngeal injury the prognosis as to life depends almost entirely on the availability of surgical care. In the absence of tracheotomy, asphyxiation occurs in almost all cases, from noninflammatory obstruction of the airway in most of them. When surgical care is available from the time of injury, few patients die from laryngeal causes. In the later stages surgical care is almost always available, and the prognosis as to life is good. When the best methods of treatment are available, the prognosis as to the airway is good in all cases in which there has been little or no loss of cartilage, though the duration of treatment may be long before permanent decannulation and fistular closure are obtained. Other things being equal, the shortest duration is observed in the cases in which the tracheotomy has been

done low. Involvement of the pharynx or of the cervical portion of the esophagus as well as of the larynx adds gravity to the immediate prognosis. If a lumen is maintained by wearing even the smallest catheter, a good swallowing tube can ultimately be obtained. Prognosis as to the voice is similar to that for the airway. So long as air passes through the larynx the patient will have at least a good loud whispered voice. When the lumen becomes adequate, the voice will develop into a "stage whisper"; later there will usually be loud phonation, rough in quality. In total atresia the voice is buccal, pharyngeal, or esophageal until a small airway is restored. In cases of total destruction of the laryngeal framework, the airway cannot usually be restored, but the pseudovoice can be developed to a useful degree, just as after laryngectomy for cancer (q.v.).

Sequelae—Formerly, wearing a cannula for life was the usual sequela of severe and often of even slight laryngeal trauma. Core-mold treatment has eliminated a large percentage of such sequelae. Deformity requiring plastic repair apart from restoration of the airway, particularly the excision of an unsightly scar, occasionally is observed. Care must be taken in such work not to create any contractile tension that may lead to stenosis. Cicatricial stenosis can usually be prevented, or if allowed to occur, it can be cured by core mold treatment (q.v.). Paralysis due to injury of nerve trunks usually remain (see "Syndromes Associated with Laryngeal Paralysis"), though a considerable degree of adjustment may develop in time (see also under "Paralysis in Relation to the Esophagus").

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CANCER OF THE LARYNX

Cancer of the larynx is a term generally applied to carcinoma only, though sometimes used for sarcoma and other more rare malignant tumors. Carcinomas may be epitheliomas or adenocarcinomas, though the former are much more common in the larynx.

Etiology.—The etiologic factors of cancer of the larynx have been expressed¹ in a formula which we may simplify as follows: $A + S + C + I + H + XYZ = E$. A stands for age, S for sex, C for senile changes in epithelium, I for irritation, H for heredity, and XYZ for the unknown factors, E stands for etiology. Age is unquestionably a factor, because the age in evidence of cancer of the larynx has, in the experience of many different investigators, shown predilection for the sixth decade of life. It is rare in a person under forty years of age, though it has been reported in teen age patients. Sex is likewise an important factor. In a series of almost 5000 deaths by cancer of the larynx reported during a four year period, it was found that 13.3 per cent of the cases occurred in women and 86.7 per cent in men. In a series of almost 700 of our own patients with malignant disease of the larynx, 92.3 per cent were men and 7.7 per cent women. In a series of 265 patients reported by Hayes Martin² 95 per cent were males. Irritation may be of many kinds but alcohol, tobacco, and vocal abuse are among the more common forms. Syphilis has been mentioned by some writers as an etiologic factor, but is now generally believed to be of no importance in causing cancer of the larynx. Martin points out that in cancer of the tongue it does seem to have some importance, in 30 per cent of his cases of cancer of the tongue there were positive Wassermann reactions. Heredity is another factor which is difficult to evaluate in relation to its importance in the development of cancer in the human being. It

is probable that cancer is not transmissible, but susceptibility to cancer and resistance to cancer are probably hereditary mendelian characteristics. As we learn more about the XYZ factors not only the cause but also the treatment of cancer should be simplified.

Pathology.—Cases of cancer of the larynx are generally divided into *intrinsic* and *extrinsic*. This terminology is based upon site of origin of the lesion. The *intrinsic* area consists of the true vocal cords, the ventricles, and the under surface of the ventricular bands. The *extrinsic* area extends from the free margins of the ventricular bands upwards, including the aryepiglottic folds and the epiglottis. It is well to define specifically these two areas, and not simply to say that intrinsic lesions are those that originate "inside the larynx" and extrinsic lesions are those that originate "outside the larynx." The behavior of the tumor is quite different in the two groups, particularly with reference to lymphatic extension, and certainly this differentiation plays a major role in determining the selection of treatment. Some laryngologists make a third classification for tumors originating in the *subglottic region*, instead of including them in the *intrinsic* group. There is certainly ample justification for paying attention to subglottic involvement, whether by origin or by extension, in connection with the selection of treatment (*q v*).

Two anatomical features have a great deal to do with the limitation of extension of carcinoma of the intrinsic larynx beyond the organ in which it originates. In the first place, the cartilaginous box formed by the thyroid cartilage offers a protection to the surrounding tissue from extension of the lesion, and in the second place, the scarcity of lymphatics in the intrinsic area plays an important role in isolating cancer originating in this region. When extension of a carcinoma does occur, it is very often due in part to the presence of infection, which causes necrosis of cartilage and destruction of the natural protection which it affords. On the other hand, in some cases neoplastic involvement of cartilage, or necrosis of cartilage by pressure, is primary, and infection secondary. Lymphatic metastasis from cancer of the larynx does occur, but in the intrinsic cases it is a late manifestation, for the above mentioned reason. What lymph vessels there are draining the intrinsic area run anteriorly, and terminate in the prelaryngeal and pretracheal nodes. The lymph

channels draining the extrinsic area flow chiefly into the upper and middle deep cervical nodes. The posterior commissure is drained downward into the party wall between the trachea and esophagus.

The *histopathology* of laryngeal tumors is extremely important, especially in relation to biopsy. In most cases the structural changes in the diseased tissues are easily determined by the section of a small specimen of tissue, but in other cases more difficulty is encountered. Invasion is the most important histologic characteristic of a malignant tumor, but of course, lawless proliferation, atypical form of cells, lack of differentiation, increased number of mitotic figures, nuclear and nucleolar changes, and hyperchromatization are among the other characteristics of malignancy. Acanthosis, hyperkeratosis, and hyperplasia may coexist with invasion and other changes constituting fully developed malignancy, but they do not necessarily signify that a lesion is malignant or will become so. In a large series of cases of cancer of the larynx seen in the author's clinic, 98 per cent were squamous or epidermoid carcinoma. This percentage is higher than it would be in the average series, because it is based on a series including an especially large proportion of cases of intrinsic and cordal cancer, which naturally constitute a larger proportion of the cases in a clinic devoted to laryngology, as compared with the proportion in a general surgical clinic or a cancer clinic. Regardless of the clinical significance of grading, it is advisable to make some effort to classify tumors according to probable degree of malignancy. Different pathologists will grade the same series of tumors a little differently, but the fundamental principles of grading are essentially the same for all. Grading is based on relative rapidity of growth, and this is determined largely by the degree of differentiation of the cells. In tumors of grade I, the cells are between 75 per cent and 100 per cent differentiated, in tumors of grade II, the cells are from 50 per cent to 75 per cent differentiated, in tumors of grade III, differentiation is between 25 and 30 per cent, and in tumors of grade IV, differentiation is noted in less than 25 per cent of the cells (Fig. 399). It has been observed by various investigators that different parts of the same tumor may show different degrees of differentiation, and hence lead to different grading. For this reason, it is important to take

specimens from several areas in the case of large growths, and in the case of smaller growths to remove as much as possible of the tumor for histologic examination, and section all parts.

Symptoms.—The symptoms of cancer of the larynx will depend on the location of the lesion. If a tumor arises on the true vocal cords, of course, hoarseness will be an early and most important symptom. If it arises in an adjacent region of the intrinsic area, as in the ventricles or on the under surface of the ventricular bands, hoarseness will be a little later developing. On the other hand, if the tumor arises in the extrinsic area, as on the upper surface of the ventricular bands, the aryepiglottic folds, or the epiglottis, hoarseness may be a very late symptom or may not develop at all. In the extrinsic area, the first symptoms are generally a sensation of discomfort, a tickling sensation, or a "lump in the throat," while a little later pain on swallowing and pain on talking may develop. The relative mildness of the early symptoms of cancer of the extrinsic larynx is very misleading, and it is the principal reason for the lateness of diagnosis of tumors of this region. As a matter of fact, even in the intrinsic cases, the hoarseness is generally considered so mild a symptom that the patient himself, unless he is a professional voice user, rarely attaches sufficient importance to it. The physician who is properly informed and aware regarding the early symptoms of cancer of the larynx will not fail to see that every case of hoarseness is thoroughly studied, with a special view to the exclusion of cancer in the diagnosis.

Later symptoms of cancer of the larynx, which may occur in either intrinsic or extrinsic cases, are dyspnea, wheezing, cough, difficulty in expelling secretions, halitosis, and cachexia. If there is complicating infection and suppuration, there will be tenderness to pressure, and in some cases even perforation of the thyroid cartilage and abscess formation. The broadening of the larynx noted on palpation is significant of advanced disease, with or without complicating infection.

Enlargement of cervical lymph nodes may be the first symptom discovered, though in the case of intrinsic lesions it is a late symptom. The intrinsic lesions most often metastasize to the prelaryngeal and pretracheal lymph nodes, and the extrinsic ones metastasize to the deeper nodes of either the upper or the middle group.

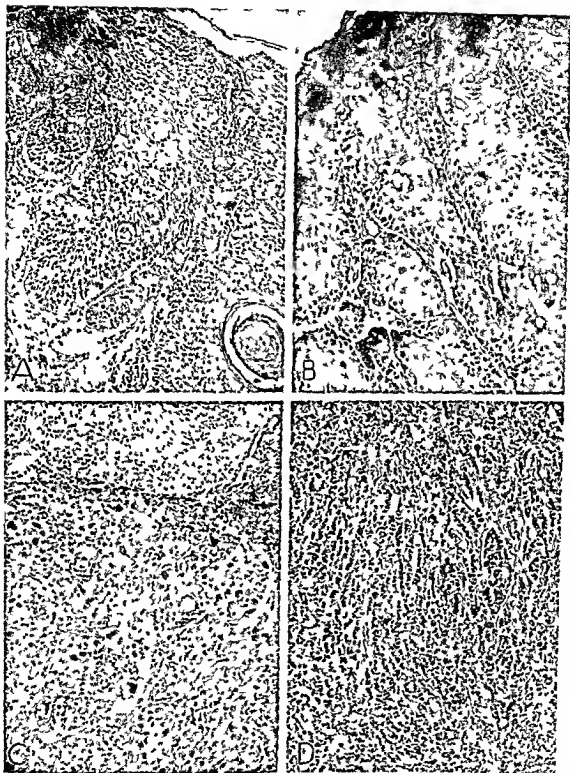


Fig. 399.—Histopathology of cancer of the larynx showing lesions of the four grades I, II, III, and IV. A, Lesion of grade I in a man forty nine years of age. This patient is still living and free of disease fifteen years after laryngofissure. B, Lesion of grade II in a man forty three years of age. Laryngectomy was performed in this case and the patient is still living and free of disease three and a half years after operation. C, Lesion of grade III in a man forty years of age. This patient was advised to have a total laryngectomy but refused and was therefore treated with protracted fractional irradiation given in two courses about six months apart but six months after the conclusion of the second course carcinoma was still demonstrable by biopsy and the patient finally consented to laryngectomy. The patient is now free of disease almost three years after operation. D, Lesion of grade IV in a woman fifty six years of age. The tissue was taken from a rather deep portion of the right ventricular band. The patient is now free of disease almost ten years after treatment by protracted fractional irradiation.

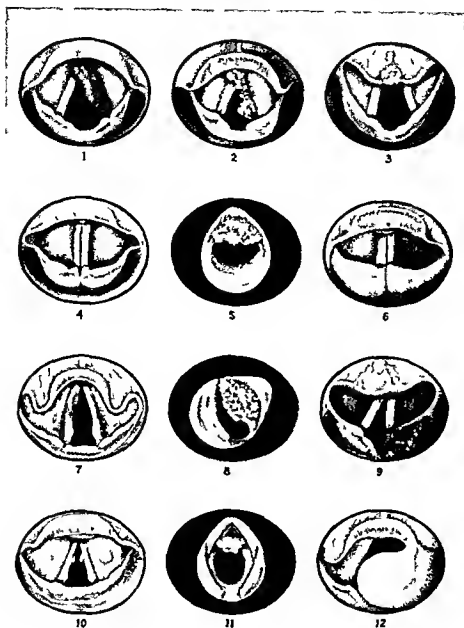


Fig. 400 —1, Carcinoma of the larynx in a man aged forty two. Almost the entire left cord is involved and the growth crosses the anterior commissure, involving the anterior end of the right cord. By the *anterior commissure technic*, the growth was removed with a normal margin of 1 cm. in all directions. To obtain this much margin it was necessary to remove the entire left arytenoid.

2, Carcinoma of the larynx in a man aged fifty six, involving the entire left cord. Laryngofissure, even by the anterior commissure technic, was contraindicated because the arytenoid region was involved and there was *fixation*.

3, Carcinoma of the central portion of the free margin of the epiglottis in a man aged forty two. There was no recurrence nine years after *direct laryngoscopic amputation* of the free portion of the epiglottis. Such removal of carcinoma of the epiglottis is justifiable only in the case of a small early lesion such as this, located on the free margin of the epiglottis and in the midline.

4, Bilateral posticus paralysis supposed to have been due to operation for "malignant goiter," though a month had intervened between the operation and the sudden onset of the paralysis. The patient, a woman aged thirty-eight, was referred to us for the fitting of a valvular cannula. Both arytenoids were tilted forward, overhanging the posterior ends of the cords. At direct laryngoscopy the arytenoids were found freely movable to closed forceps pressure ("passive mobility test"). When the direct laryngoscope was passed into the hypopharynx posterior to the larynx a carcinoma was found at the orifice of the cervical esophagus (cf. 5).

Laryngeal Appearances.—The gross appearance of cancer of the larynx varies greatly. In some early cases it is seen as simply a slight thickening and roughening of the cordal surface. In other cases there is the whitish "cauliflower" appearance so often characteristic of carcinoma. In still other cases, the appearance is that of a superficial ulceration. Sometimes the color is whitish, sometimes red. Impairment of cordal motility is sooner or later produced in the cordal lesions, as the tumor infiltrates the muscles. Motility should always be closely observed, noting especially the behavior of the cord on abduction, following phonation. Particular care should be taken to examine the subglottic region. This is best done on extreme abduction. If there is any suspicion at all with regard to the subglottic region, direct laryngoscopy should be done, using the smooth-tipped anterior commissure laryngoscope (Fig 400).

Diagnosis.—That the diagnosis of cancer of the larynx be made early is vitally important, because the larynx is a part of the body in which cancer diagnosed early can be cured in a high percentage of cases, whereas the same patients who could be cured if diagnosis were made

early will become incurable, or curable in a much lower percentage of cases, if the diagnosis is made late. Hence the great importance of emphasizing the need to pay attention not only to hoarseness but to the other less obvious symptoms referable to the throat. The public and the profession alike should be impressed again and again with this fact.

In the presence of hoarseness or other symptoms referable to the throat a systematic study is indicated, consisting of a careful history, examination of the throat with the tongue depressor and laryngeal mirror, careful palpation of the neck, a complete general physical examination, and very often roentgen-ray examination of the chest, serologic test, and other laboratory studies. In cases in which the larynx and piriform sinuses cannot be perfectly visualized by means of the laryngeal mirror, direct laryngoscopy is indicated. Complete visualization of the interior of the larynx, right up into the anterior commissure, must be obtained. "Death often lurks under an overhanging epiglottis" in the form of a carcinoma involving the anterior portions of the vocal cords. Direct laryngoscopy is also indicated for supplementary inspection and the taking of tissue for

5, Same patient as at 4. The long direct laryngoscope, passed into the hypopharynx and cervical esophagus, revealed a carcinoma that evidently had produced the paralysis by involvement of both recurrent nerves. Histologically this growth was an adenocarcinoma as was the antecedent goiter. The esophageal growth recurred about a year after disappearance under irradiation.

6, Edema of both aryepiglottic folds in a woman aged fifty-six. Biopsy from one of these folds had been negative for both malignant disease and tuberculosis before admission. Endoscopy with the laryngoscope revealed an ulcerating fungating lesion on the hypopharyngeal wall of the larynx. Evidently the edema was part of the perimalignant inflammatory zone not yet invaded by the cancerous process. Irradiation arrested the growth. Recurrence and metastases were fatal in the fifth year afterward.

7, A cupped but not ulcerated squamous cell carcinoma of the right vocal cord. The growth seemed limited to the cordal margin, but direct laryngoscopy revealed extension into the ventricle and into the subglottic region (cf 8).

8, Same patient as shown at 7. Lifting of the right ventricular band with the lip of the direct laryngoscope drawn upward to the right reveals the actual extent of the lesion. What seemed in the mirror to be a small growth on the edge of the cord was really only the margin of a lesion that extended outward on the ventricular floor, and the dragging upward brought into view an extensive subglottic involvement that was invisible in the mirror. The extent of the lesion thus revealed called for total laryngectomy.

9, Ulcerating fungating extrinsic carcinoma of the larynx in a woman aged sixty-five. A negative opinion had been given by a laryngologist after mirror examination about two months previously. Doubtless the growth at that time had not emerged from its primary focus in the hypopharynx.

10, Mirror appearance in the case of a woman aged twenty-seven, sent in for the removal of two masses of papilloma, one on each vocal cord, a diagnosis not unwarranted tentatively on reflected appearances alone. Direct laryngoscopy, however, showed there was only one mass and it looked malignant rather than benign (cf 11).

11, Schematic presentation of view through the direct laryngoscope in the same patient as shown in the preceding illustration. What seemed, in the mirror, to be two separate growths on the cords is seen to be a continuous growth across the anterior commissure in the subglottic region. Biopsy revealed a squamous-cell carcinoma. The patient was alive and well when last seen five years after laryngofissure done by the anterior commissure technic.

12, Carcinoma probably originating in the sacculus laryngis of a woman aged fifty. The left aryepiglottic fold and left ventricular band are obliterated in one mass that appeared to have sprung from the region between the cricoid cartilage and the left wing of the thyroid cartilage. Superficial biopsies had been negative, but when a specimen was obtained from a depth of about 1 cm a diagnosis of "cystic adenocarcinoma" was made. The patient is still free from recurrence eight years after a course of protracted fractional irradiation.

biopsy, if a lesion suggestive of neoplasm is seen in the mirror

In recent years the roentgen-ray examination has become of increasing value in connection with the diagnosis of laryngeal disease. The technic of roentgen ray study in the lateral projection, both without and with an opaque mixture, has been perfected for some time, but only quite recently has the development of *planigraphy* (or *tomography*) made it possible to visualize the even more important coronal plane view, in which we may study the right and left sides separately. This method demonstrates beautifully the contours of the ventricles and the subglottic region. The visualization of this latter region is perhaps most often of definite practical value (Fig. 349). Of course, even the finest roentgen ray technic does not give us the histopathologic structure of the lesion, and the overenthusiastic roentgenologist should be discouraged from trying to go too far in his interpretation of the roentgenographic appearances. One of the pioneers in the study of the larynx by "tomography" was Felix Leborgne of Monte video, who recently published the results of his experience in the roentgen ray study of cancer of the larynx in a beautifully illustrated book.³

Differential Diagnosis—In the first place, the location of the lesion affords a good start in differential diagnosis. The anterior two thirds of the length of the vocal cords is for some reason much more commonly the site of development of a carcinoma than the posterior third, and the posterior commissure is almost never primarily invaded. On the other hand, though *tuberculosis* very commonly affects the posterior commissure, it does also affect the cord, and in fact one characteristic type of appearance in tuberculosis is the reddening and slight thickening of one cord. In tuberculosis there is more commonly an associated generalized edema affecting very often the epiglottis, producing the "turban" epiglottis, and generally affecting also the aryepiglottic folds. *Syphilis* can scarcely be said to have a characteristic appearance, but it should always be thought of, and the possibility of its presence eliminated by a serologic test. The positive serologic reaction does not, of course, prove that the patient does not have cancer in addition to syphilis. In some cases we have found syphilis and cancer in the same patient, and both affecting the larynx, in at least one of our cases syphilis, cancer, and tuberculosis were all three coexisting. **Biopsy is**

the final arbiter in such cases, and the specimen is generally most accurately taken by direct laryngoscopy. Occasionally, however, especially in the extrinsic cases, it may be just as convenient to take the specimen by indirect laryngoscopy, using a curved biopsy forceps. *Benign tumors* can generally be differentiated by their gross appearance in the laryngeal mirror. *Polyps*, *organizing hematomas*, and *vocal nodules* have a smooth surface and are very often pedunculated. *Carcinomas* generally present a roughened surface, and are very rarely pedunculated. *Papilloma* generally has a roughened surface and is pinkish in color, and not infrequently shows close resemblance to carcinoma. Its multiplicity is one differentiating characteristic. *Leukoplakia* and *keratosis*, *pachydermia*, and other so called *precancerous* conditions call for thorough examination and continued observation. *Recurrent laryngeal paralysis* and *cricothyroid arthritis* have not infrequently been mistaken for carcinoma, and vice versa. Mere impairment of motion should not mislead one into making either of these diagnoses without very careful scrutiny, using the direct method if necessary to supplement the indirect. Among the more rare conditions from which carcinoma is to be differentiated might be mentioned *eversion of the ventricle*, *blastomycosis*, *scleroma*, *amyloid disease*, *neurofibroma*, *chondroma*, and, of course, the more rare forms of malignant tumors such as *sarcoma*.

Treatment—In a general way, it may be said that the treatment of cancer of the larynx is by *surgery* or by some form of *irradiation* or by a combination of these methods. In the surgical treatment, the choice lies between *total laryngectomy* and some form of *partial laryngectomy*. The most commonly used form of partial laryngectomy is that known as *thyrotomy* or *laryngofissure*, in which access is had to the growth by a splitting of the thyroid cartilage, after which the growth is removed along with a margin of normal tissue. Another form of partial laryngectomy is *hemilaryngectomy*, an operation which has given good results in the hands of some operators, particularly Hautant.⁴ Occasionally *epiglottidectomy* is justifiable in the case of tumors limited strictly to the epiglottis. *Endoscopic resection* under suspension laryngoscopy has been practiced by Lynch, Lejeune, New, and a few others, but certainly this technic is suitable only to very early and limited cordal lesions. *Total laryngectomy* may be done by

various techniques, and some of them differ so much as to constitute really different operations. If the entire epiglottis and the entire cricoid are not removed, as they need not necessarily be in every case, perhaps we should call the operation *subtotal*. On the other hand, some operators always remove the hyoid bone and pre epiglottic space, which makes the operation something more than a total laryngectomy. In the case of extrinsic lesions, with involvement of

mmistered by some form of "protracted fractional" (Coutard) technic, or the use of some form of radium, as for example the "saturation technic," with radium packs applied to the neck externally. In certain cases radon (gold "seeds" containing radium emanations) or removable radium needles are inserted. In the "window resection" technic of Douglas Harmer radium needles were laid in a window made in the thyroid ala. One of the forms of combined

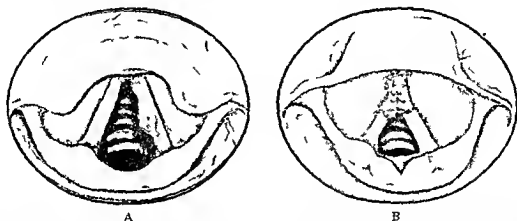


Fig. 401 —Mittor appearance of lesions suitable for *laryngofissure*. A Lesion involving the middle third of the cord, for which the clipping operation would be used. B Lesion in the anterior commissure which would require the use of the 'anterior commissure' technic.

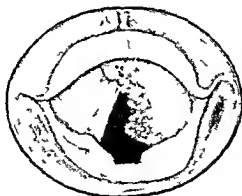


Fig. 402 —Lesion which has reached not only the anterior but the posterior extremity of the cord, and also produced some impairment in cordal motility. *Laryngectomy* is the treatment of choice in such a case.

adjacent structures (as, for example, the pharynx or the base of the tongue), obviously an attempt at surgical extirpation will require more than total laryngectomy. In such cases few operators feel favorable to surgical treatment, but those who do generally use the technic of *laryngopharyngectomy* of Hayes Martin or the lateral transthyroid procedure of H. Boylan Orton (p. 180).

Treatment by irradiation may mean the use of roentgen rays, which are now generally ad-

ministry and irradiation was the technic of application of radium through a laryngostomy opening formerly used by Martin² and still applicable in certain cases. Another form of combined surgery and irradiation is the neck dissection and radon implantation used in the treatment of cervical metastasis.

Selection of Method of Treatment—The methods of treatment of cancer of the larynx which the author⁶ uses routinely are *laryngofissure* by either the "clipping" or the "anterior

commissure' technic, *laryngectomy*, and *irradiation by roentgen rays*. The major considerations in the selection of a method of treatment are

first, the prospect of cure of a fatal disease and, second, conservation of function, in other words, voice. Among the secondary consider

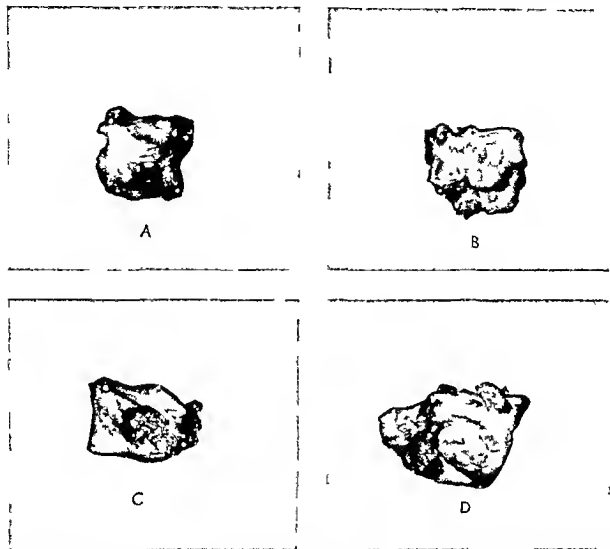


Fig 403 —Operative specimens showing early carcinoma removed by laryngofissure with a margin of normal tissue. A Squamous-cell carcinoma grade II limited to middle third of left vocal cord. Extirpation accomplished by laryngofissure clipping technic. B Squamous-cell carcinoma grade II springing from anterior portion of left vocal cord. Excision accomplished by clipping laryngofissure. C, Squamous-cell carcinoma grade I involving left cord and showing slight tendency to extend subglottically. Excision accomplished by clipping laryngofissure. D Squamous-cell carcinoma grade II involving almost the entire right cord and the anterior portion of the left. Excision accomplished by laryngofissure using the Jackson anterior commissure technic.

Fig 404 —Operative specimens in cases of cancer of the larynx in which total laryngectomy was done. A Carcinoma grade II involving right cord and extending subglottically. Some impairment of motility was present in this case. B Larynx with tumor said to be spindle squamous-cell carcinoma grade III to IV (Patient has had no recurrence in over four years.) C Squamous-cell carcinoma grade II involving left cord and showing some subglottic extension. D Squamous-cell carcinoma grade II involving anterior two-thirds of both cords and extending subglottically. E, Extensive squamous-cell carcinoma grade II extending up into the base of the epiglottis and also subglottically causing sufficient obstruction of the airway that tracheotomy had been required prior to laryngectomy (Patient still free of recurrence or metastasis three years after operation.) F, Squamous-cell carcinoma grade III involving the left true and false cord. Cervical metastasis developed which was treated by neck dissection and radon seed implantation.

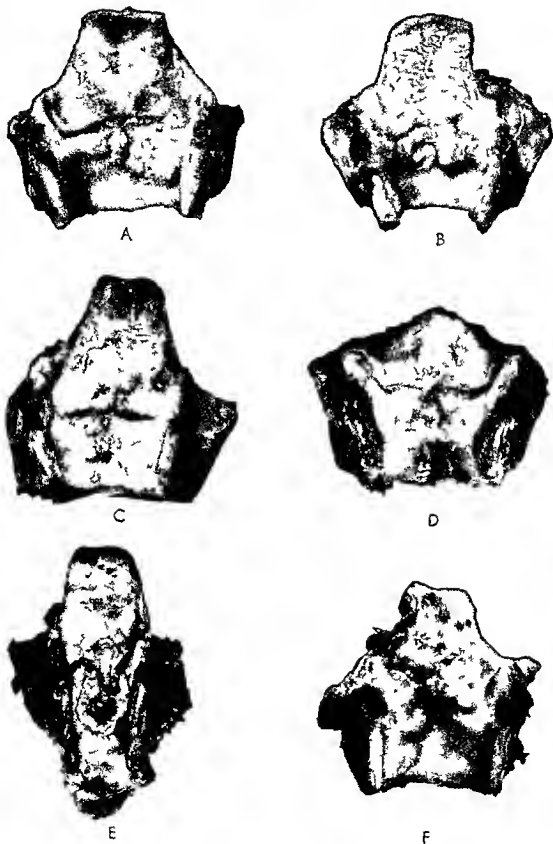


Fig 404

Description on page 566

ations are duration of morbidity, degree of discomfort, and possibility of complications. Our criteria for the selection of a method of treatment are (1) the presence or absence of cervical metastases, (2) the location and extent of the lesion, as evidenced by its appearance on direct and indirect laryngoscopy and in lateral and planigraphic roentgenographic studies, (3) the motility and mobility of the laryngeal structures, (4) the histologic character of the lesion and (5) the general physical condition and temperament of the patient. 'Presence or absence of cervical metastases' is put first, because if cervical metastases are present, regardless of all other considerations, the case falls into the group of cases which generally should have irradiation by roentgen rays as the first treatment

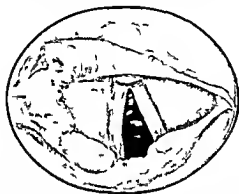


Fig 405—Lesion involving right aryepiglottic fold and right side of epiglottis. In *extrinsic lesions* of this kind surgery offers little prospect of cure even by radical procedures but irradiation may be effective

rather than any form of partial or total laryngectomy, such irradiation to be followed by neck dissection and interstitial irradiation

INDICATIONS FOR SURGICAL TREATMENT—(1) Lesions occupying the middle third of one vocal cord are suitable for laryngofissure by the 'clipping' technic (2) Lesions reaching the anterior commissure and even involving the opposite cord are also amenable to extirpation by the laryngofissure route, but in such cases the 'anterior commissure' technic should be used (Figs 401 and 403) (3) Lesions in which the growth is cordal but has reached the posterior end of the cord and produced impairment of motility, or has extended subglottically, ordinarily call for total laryngectomy (Fig 402), as do lesions of the ventricles and the ventricular bands (4) Lesions in which tumor has invaded cartilage (but not the overlying muscles) also

call for laryngectomy, provided there are no metastases (Fig 404)

INDICATIONS FOR IRRADIATION—(1) Lesions unsuitable for laryngofissure and for which laryngectomy is contraindicated by the age, physical condition, or temperament of the patient. These factors rarely, if ever, contraindicate the less radical procedure of laryngofissure, but they are to be regarded as relative contraindications to laryngectomy (2) Growths "inoperable" because extrinsic by origin or extension (Fig 405) or because of cervical metastases. The latter cases must be treated by protracted irradiation, followed immediately by implantation of radon, either through the skin, or better, after surgical exposure. Cervical metastasis developing after operation, without local recurrence, should be treated by neck dissection and radon implantation, not necessarily preceded by deep roentgen ray therapy (3) Lesions which have reached the posterior extremity of the cord but have not produced impairment of motility. Such growths constitute a group for which laryngofissure is contraindicated but for which irradiation may be preferred to laryngectomy. It is the consensus at present that impairment of motility contraindicates laryngofissure, but that it is not a contraindication to irradiation. As a matter of fact, it is not a favorable sign for irradiation either, and complete fixation of one or both sides of the larynx is almost a contraindication

There are those who minimize the importance of the *histopathologic picture*, but there can be no doubt that the ultimate prognosis will be most favorable in the better differentiated, slower growing, lower grade tumors, regardless of the method of treatment. Furthermore, 'grading' should be taken into consideration in the selection of treatment, especially in the case of borderline lesions. In a general way it may be said that in the less differentiated, "higher grade" tumors one should feel inclined toward more radical surgery or irradiation, though with regard to selection of irradiation, it should be pointed out that the tumors of grade I and grade II, which used to be considered *radio-resistant*, are more apt to be radiocurable than the less differentiated tumors of 'higher grade'. These latter tumors, of grade III and IV, carry a poor prognosis with any method of treatment

Surgical Treatment—**LARYNGOFISSURE**—'Laryngofissure' means simply splitting of the larynx, and for this reason, some writers object

to the application of the term to the operation for removal of a part of the larynx for early cancer (Fig 406) (Synonyms are *partial laryngectomy*, *thyrotomy*, and *thyrochondrotomy*) Space does not permit an account of the very interesting history of this operation, but a good account of it may be found elsewhere.^{1 7}

Technic—The technic of laryngofissure used by the author at the Temple University Clinic at the present time is as follows

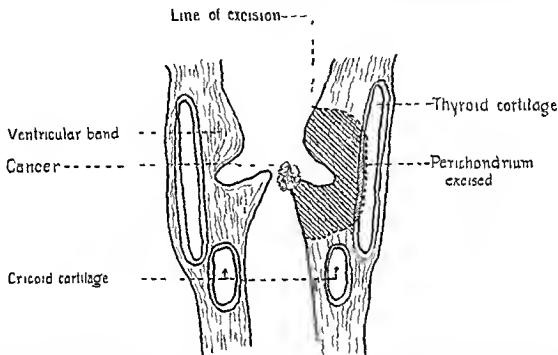


Fig 406—Schematic representation of the operation of laryngofissure in coronal section. Note the small tumor on the edge of the cord and note how the operation permits removal of internal perichondrium and overlying soft tissue of the cord, ventricle, and ventricular band. In the case of very limited cordal lesions, the removal of the ventricular band may not be necessary.

Step 1 Anesthesia Local anesthesia by infiltration with novocaine (1 per cent) intradermally and then subcutaneously along the midline of the neck is used in all cases. The local anesthesia is completed by topical application of cocaine (10 per cent) or pontocaine (2 per cent) to the interior of the larynx and upper trachea on a swab introduced through an incision in the cricothyroid membrane after the operation is under way. This application is analgesic, and eliminates the cough reflex from the larynx and upper trachea, also, it produces slight ischemia of the mucous membrane and soft tissue of the interior of the larynx, lessening the amount of oozing from cut mucous membrane and muscles. General anesthesia, while entirely unobjectionable for total laryngectomy, is, in the author's opinion, contraindicated for laryngofissure, because it greatly increases the operative risk, both from hemorrhage and from pulmonary complications.

Step 2 The Incision A straight vertical midline incision from the thyroid notch to just below the level of the cricoid is used. If tracheotomy is to be done, the incision may be prolonged downward to the suprasternal

notch, but we do not do tracheotomy as a part of the standard operation. Chevalier Jackson has emphasized the importance of not skeletonizing the larynx in laryngofissure, because of the unnecessary trauma thus inflicted on the external perichondrium and consequent impairment of nutrition of the thyroid alae.

Step 3 Special Cover As soon as the incision has been made, the thyroid and cricoid cartilages exposed, and hemostasis accomplished, a special cover consisting of a sheet with a slit in it to fit the wound is put in place, the edges of the opening being clipped to the skin margins. This cover helps to maintain scrupulous clean-

liness during the operation, and particularly during the manipulation of the cartilage.

Step 4 Incision of Cricothyroid Membrane After placement of the special cover, the cricothyroid membrane is incised either transversely or vertically, and topical application of cocaine made to the interior of the larynx. At first a few drops are squeezed out by pressure of the sponge against the borders of the incision, and then the sponge itself is gently introduced. Several successive applications are made in the same way.

Step 5 Division of the Thyroid Cartilage When the lesion does not reach the anterior commissure, the "clipping" technic is used, dividing the thyroid cartilage exactly in the midline with shears, but in cases where the lesion does reach the anterior commissure, the special technic for such cases first described in 1922 by Chevalier Jackson¹ is preferable. The essential feature of this technic is division of the thyroid cartilage from the outside by means of an electrically-driven circular saw, or in the case of patients with a very soft cartilage, a knife, this incision not going clear through the internal perichondrium. In this way one avoids cutting through

the tumor In a recent series of fifty cases⁷ the clipping technic was used in thirty two and the Jackson anterior commissure technic in eighteen

Apropos of anterior commissure lesions, mention should be made of an interesting study

the attachment of the true vocal cords to the thyroid cartilage anteriorly, and because of its structure Broyles advises the removal of a portion of each thyroid ala along with the anterior portion of the cords in the anterior commissure



Fig 407 —The operation of laryngofissure The cricothyroid membrane has been incised preliminary to the division of the thyroid cartilage Cocaine (10 per cent) or pontocaine (2 per cent) is applied topically through this incision in order to anesthetize the interior of the larynx and the upper trachea The next step would be the division of the thyroid cartilage by means of shears if the clipping technic is used or by the circular saw if the anterior commissure technic is used

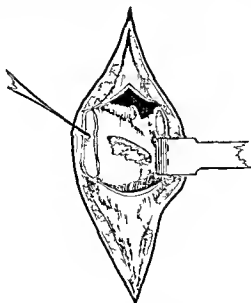


Fig 408

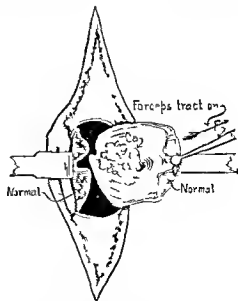


Fig 409

Fig 408 —View of interior of larynx obtained in laryngofissure by the clipping technic.

Fig 409 —Schematic representation of laryngofissure by the anterior commissure technic Note that soft tissues are cut first on the less involved side and that the flap is then reflected in such a way as to permit inspection of the lesion before the excision is completed

by Broyles,⁹ in which the 'anterior commissure tendon' is described This 'tendon' constitutes

cases This procedure has also been advised by Clerf¹⁰

Step 6 Inspection of Interior of the Larynx If the patient has been properly studied beforehand by direct as well as indirect laryngoscopy, one should have a fairly accurate idea of the location and extent of the lesion, but after the larynx is opened it should again be carefully examined before the excision is begun. In the clipping operation this is simple but in the anterior commissure operation it is a little more difficult. In the former, we can inspect the lesion before doing even the subperichondrial dissection (Fig 408) but in the anterior commissure operation it is necessary first to separate off the internal perichondrium on both sides and then to section vertically the *less involved cord and ventricle* so that it can be reflected in such a way as to permit good visualization of the entire interior of the larynx. This careful inspection permits one to decide upon the extent of excision necessary to give a safe margin of normal tissue around the growth (Fig 409).

Step 7 Partial Removal of Cartilage We do not resect the cartilage but some operators have thought it desirable to do this (Lack advocated resection of the thyroid ala along with the soft tissue bearing the growth, because he believed that it was easier to be sure of adequate removal in this way. St. Clair Thomson¹¹ made it a part of his technique to do a subperichondrial resection of the thyroid ala before excising the tumor and adjacent soft tissue.)

Step 8 Excision of the Growth After subperichondrial dissection, the tumor bearing mass is excised with special curved scissors (Fig 410) including generally the tip of the vocal process of the arytenoid on the more involved side. If possible, the mass excised is kept in one piece, but of course it may be necessary to remove additional tissue if it is found that the margin is insufficient

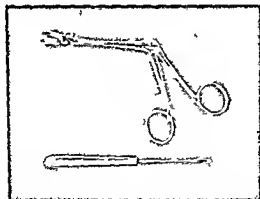


Fig 410—Special curved scissors (right and left) designed by Chevalier Jackson for intralaryngeal surgery, particularly laryngofissure. Below, perichondrial elevator for use in laryngofissure.

on one side or the other. To take a full centimeter of normal tissue on all sides according to the 'centimeter rule' of Chevalier Jackson, is ideal but not always practicable (Fig 403).

Step 9 Hemostasis Plain or iodoform gauze is tucked into the open larynx immediately after the excision, while the operative specimen is inspected, and most of the bleeding will generally stop with this alone. If a spurting vessel can be caught quickly with a hemostat, so much the better, because later it may retract and be difficult to find. The larynx must be got absolutely dry

before closing or postoperative bleeding will be sure to occur. Occasionally one or more suture ligatures of fine catgut are used, particularly in the region of the arytenoid cartilage. If the excision has included one or both ventricular bands the base of the epiglottis will be left without support and should be stitched forward as suggested by Gabriel Tucker.¹² This will tend to prevent stenosis anteriorly.

Step 10 Closure No sutures are put through the perichondrium or cartilage, though a suture or two may be used in closing the incision in the cricothyroid



Fig 411—Postoperative appearance of neck at time of patient's discharge from the hospital (eighth to tenth day) after laryngofissure. The wound is closed without drainage and in most cases heals by first intention.

membrane. The fascia and muscles are closed with buried sutures (alloy steel wire) and the skin with Michel's clamps. We have used no drainage for the past several years though formerly a small wick of iodoform gauze was left in the lower end of the wound. The older practice of leaving the wound open and packing down to the cartilage has been entirely abandoned.

Step 11 Dressing A plain gauze dressing is applied. The dressings are changed daily, and the clips removed on the third or fourth day.

After-Care—The patient is kept sitting up, or rather with the head of the bed elevated. He is instructed not to talk, but to use a pad and pencil to express himself. After a few hours he is given liquids and soft solids, being encouraged to sit up, lean slightly forward and take a good swallow rather than sipping or taking the liquid through a glass tube. In very few of our cases has a feeding tube been used, but if an extensive excision is made, and especially if a large portion of the arytenoid is removed, it may be necessary to insert one. Opiates are contraindicated.

cated, and only the very mildest sedation is permissible. The wound is generally healed externally in four or five days, but the healing of the interior takes several weeks. Most patients are discharged from the hospital in from one week to ten days (Fig 411).

Complications of Laryngofissure—*Hemorrhage*, if it occurs, generally occurs within the first twelve hours. It was observed in only two cases in a recent series of fifty.⁷ To these it was necessary to pack the larynx temporarily, but generally the bleeding point can be found and the vessel ligated. Packing seemed to have no harmful effects, though in one of the cases there

Abscess, perichondritis, and chondral necrosis occur in a certain percentage of cases and may require drainage. Often the extrusion of a little sequestrum of cartilage or bone will be followed by prompt healing, but in some cases a number of pieces come away before the wound will heal. This complication should be treated conservatively.

Bronchopneumonia occurred in only one case of the previously mentioned series, though pulmonary complications (*pneumonia, atelectasis, pulmonary abscess*) were formerly rather frequent and often fatal, in patients who had undergone laryngofissure. The use of a local anesthetic and

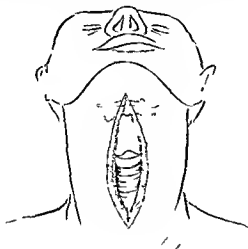


Fig 412

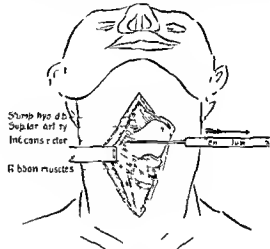


Fig 413

Fig 412—Incision for laryngectomy. A simple vertical midline incision from the hyoid bone to the supra-sternal notch. If desired, a separate circular incision may be made for the tracheal stoma, as indicated by the dotted line. This permits conservation of a strip of skin between the tracheal stoma and the lower end of the vertical incision.

Fig 413—Skeletonization of the larynx. Note that ribbon muscles are retracted laterally, the larynx is rotated laterally by means of a small hook tenaculum, and inferior constrictor fibers are about to be cut along the posterior border of the thyroid ala (Step 4). The central portion of hyoid bone has already been resected, though this may be left until after division of constrictor muscle, as described in the text.

was an especially active growth of granulations, requiring direct laryngoscopic removal. Obviously, it is necessary to insert a tracheal cannula if packing is done. As has been stated, the risk from postoperative hemorrhage is much greater if general anesthesia is used, than with local anesthesia.

Granulomas form in about 35 per cent of the cases. Generally they shrink and disappear in a few weeks. They should be given a chance to do that, and if they do not, they should be removed by direct laryngoscopy, because their presence, even though not obstructive to an important degree, will interfere with the development of voice.

the withholding of postoperative opiates are probably the chief factors in the lowered incidence of pulmonary complications.

LARYNGECTOMY—A full outline of the history of laryngectomy is given elsewhere.¹

Preparation—Proper attention to preoperative study and treatment will have a profound effect on operative morbidity and mortality. Thorough physical examination, supplemented by roentgen-ray examination of the chest, serologic study, blood count, urinalysis, and blood-chemistry determination are indicated. Other conditions found may not change the course of treatment necessary for the cancer of the larynx, but they often call for certain preliminary treat-

ment *Oral sepsis* must be eradicated even if it takes two weeks or longer to do so. If there is sugar in the urine or the blood sugar is above normal, an effort should be made to correct or improve this condition before operation.

Technic—The earlier operators used what Chevalier Jackson¹³ has called a *wide field* operation as compared with the *narrow field* types of operation that have come to be preferred in recent years. Of course, in cases where there is extension outside the larynx, a "wide field" type of operation is necessary, if surgical extirpation is to be attempted, but in the intrinsic cases, a "narrow field" type of operation is to be preferred, because the narrower the field, and the greater the conservation of normal structures, the less will be the risk and the shorter the hospitalization. The steps in laryngectomy as we are now doing it are as follows:

Step 1 Anesthesia Infiltration of the skin and tissues of the neck with novocaine (1 per cent) or cervical block with a basal anesthesia of avertin by rectum supplemented by intravenous sodium pentothal, is used in the author's clinic, though Martin,² New,¹⁴ Cunnings¹⁵ and others prefer to use local only. If there is any respiratory obstruction a rubber nasal airway should be inserted or if the obstruction is glottic or subglottic an intratracheal catheter or a bronchoscope may be used.

Step 2 Incision A midline incision is made from the hyoid bone to the suprasternal notch (Fig. 412). The incision should not be continued downward below where it is desired to locate the stoma, as this causes an unnecessary downward extension of the scar. If desired the vertical incision can be made still shorter, and a circular "buttonhole" opening made for the tracheal stoma, leaving a narrow band of skin between the lower extremity of the vertical incision and the circular one (Fig. 412). This technic is particularly desirable in cases where there may be sloughing as in patients who have had extensive irradiation.

Step 3 Division of Thyroid Isthmus As soon as the skin and fascia are incised, the thyroid isthmus is exposed just below the cricoid cartilage and divided between Kelly clamps. The cut ends are sutured with chromic catgut and the clamps removed. This procedure exposes the anterior surface of the trachea and makes it possible to let air in at any moment if respiratory obstruction develops.

Step 4 Skeletonization of the Larynx The sternohyoid muscles as well as the sternothyroids and thyrohyoids are separated in the midline, and the insertions of the latter two into the thyroid cartilage separated from the thyroid cartilage by subperichondrial dissection so that if possible, their continuity with each other is preserved (Fig. 413). The inferior constrictor muscles of the pharynx are then divided along the posterior edges of the thyroid alae, and the other muscular attachments severed, the inferior laryngeal vessels being ligated as they are reached. This skeletonization is carried out first on one side and then on the other, or the surgeon may do one side and have an assistant do the other at the

same time, as suggested by de Sanson Crowe and Broyles¹⁶ have described a technic according to which the entire skeletonization is done subperichondrially. This might be considered an extreme form of the "narrow field" (Jackson) or "close dissection" (Gatewood) technic. These names describe a general trend toward a more conservative technic of operating for the extirpation of the larynx in cases of intrinsic cancer, rather than any one individual's personal technic. Experience has shown that the anatomical basis for the use of the "narrow field" type of operation in such cases is sound. At this point it should be stated, however, that the "narrow field" technic is not to be followed in cases where the lesion has extended through the thyroid cartilage. In such cases, obviously the overlying muscle should be left attached to the larynx and removed along with it, or removed first. Hayes Martin² prefers always to remove the inner ribbon muscles (sternothyroid and thyrohyoid) with the larynx, but leaves the sternohyoid muscles intact for overlapping in closure.

Step 5 Division or Resection of the Hyoid Bone The larynx can be removed without disturbing the hyoid

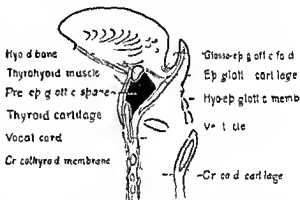


Fig. 414—Schematic sketch of sagittal section of the normal larynx, showing pre-epiglottic space (of Boyer) and hyo-epiglottic membrane which separates it from the base of the tongue. This space is early invaded by cancer of the ventricular bands and base of the epiglottis.

bone, but most operators feel that division (New¹⁴) or resection (Orton,³ Cunnings¹⁵, Clerf¹⁷) is of advantage. Facilitation of pharynx closure is the reason most often given for this procedure, but another good reason for partial or complete resection is that if this is done complete removal of the pre-epiglottic space is more easily accomplished (Biss¹⁸) and this is important, especially in cases in which the lesion involves the ventricles or ventricular bands, because it has been shown (Leroux-Robert¹⁹) that such lesions readily extend to this space (Fig. 414). It is the author's practice, in most cases, to resect the central portion of the body of the hyoid bone, but removal of the entire bone is useless, and necessitates the detachment of many muscles from their insertions into it. Martin prefers always to leave the hyoid bone undisturbed.

Step 6 Amputation of the Trachea The trachea is divided just below its first ring, and stitched forward with one temporary suture to the skin. An inner cannula wrapped with petrolatum gauze is inserted into the trachea to prevent blood from running down into it as

the separation of the posterior surface of the larynx from the esophagus and pharynx is carried upward. It has been suggested (Babcock²⁹) that whenever possible the amputation be made through the cricoid cartilage in order to obtain a more rigid stoma that may be less likely to require the permanent wearing of a cannula. Disadvantages of this procedure are that the line of incision may come too close to the borders of the lesion (in cases where there is any subglottic extension) and that there is apt to be considerably more bleeding, requiring delay for hemostasis.

Step 7 Cutting of Cornua and Ligation of Superior Laryngeal Arteries As the larynx is lifted upward and the superior cornua of the thyroid cartilage are reached, they are amputated or dissected free. If they are to be amputated and left in situ care must be taken not to

dividing the pharynx with one simple stroke of the knife, along the upper concave surface of the clamp. This prevents contamination of the wound with pharyngeal contents, and simplifies suture of the pharynx. In order not to include a portion of the epiglottis in the clamp, it is necessary to pull the epiglottis down into the supra-glottic larynx with a hook. The author has found that this maneuver is more easily accomplished if the larynx is split through the posterior commissure with shears (Figs 415 and 416).

Step 9 Closure of the Pharynx If the Brazilian clamp is used, first a temporary continuous suture of black silk is inserted, and then after removal of the clamp, a continuous inverting suture of no. 00 chromic catgut. The edges of tissue which have been compressed by the clamp are thus inverted.

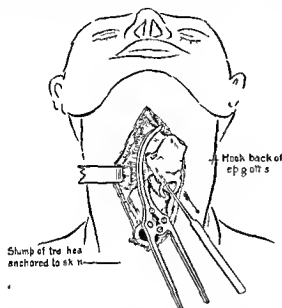


Fig 415

Fig 415—Application of Vasconcelos Barretto clamp to pharynx before opening it. This permits amputation of the larynx by a relatively aseptic technic, since the pharynx is never opened into the wound (Step 8).

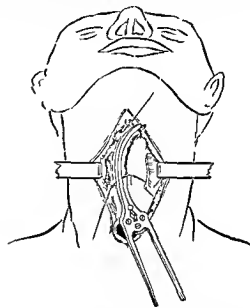


Fig 416

Fig 416—The larynx has been removed by cutting the pharynx along the upper concave surface of clamp. A temporary suture of black silk has been introduced and the clamp is about to be removed so that suture of pharynx can be completed with Cushing stitch of chromic catgut (Step 9).

denude them of perichondrium. If they are dissected free, it must be borne in mind that their tips are just submucosal and care taken not to produce a perforation. Immediately after amputating the cornua, or dissecting them free, the superior laryngeal vessels should be looked for, and ligated when found. They are ordinarily seen entering the thyrohyoid membrane just a little above the edge of the thyroid cartilage and a little anterior to the cornua.

Step 8 Opening of the Pharynx and Removal of the Larynx The pharynx can now be opened in the midline posteriorly, just above the arytenoids and posterior commissure, the incision being continued around both sides to the valleculae, care being taken to conserve as much mucous membrane as possible. However, an innovation in technic suggested by two Brazilian surgeons (Vasconcelos and Barretto²⁹) greatly facilitates this part of the operation. This consists in the application of a special clamp to the pharynx before it is opened, then

If the clamp is not used, it is necessary to locate and close the upper corners or upper extremity of the pharyngeal wound with great care, and then to complete the closure in the form of a 'U' or a 'Y,' or simply a horizontal or vertical line. Chromic catgut no. 00 or no. 000 interrupted sutures are used, and at least every other one, if not all, should be submucosal. This first line of sutures is reinforced by two more (chromic catgut no. 00 or alloy steel no. 35) which include the remaining portions of the pharyngeal constrictor muscles, and the perichondrium if dissection has been subperichondrial.

Step 10 Closure of the Muscles The sternohyoid muscles, which have been preserved intact, and also the sternothyroid thyrohyoid muscles if they have been preserved are brought together and overlapped in the midline (Fig 417). This gives firm support to the pharyngeal suture line. Additional support is given by a 'corset' (Tappa) of perichondrium if subperichondrial dissection has been done.

Step 11 Drainage Elaborate drainage and even irrigation was formerly the routine procedure in laryngectomy, but the improvements in technic of recent years have made such methods obsolete. The author has used with great success a simple technic of drainage by one small (Babcock) elbow sump tube of glass or alloy steel inserted under the muscles into which is inserted a small catheter connected to a little (flea power) electric pump (Fig. 417). Thus gentle negative pressure is applied for the first thirty six to forty eight hours after which the sump is generally removed. By this means a considerable quantity of serosanguinous fluid is drained off and thus not only is wound tension reduced but substance which would make good culture medium for wound infection is eliminated.

Step 12 Closure of Skin The skin margins are approximated to the mucosal edges of the tracheal stump and to each other in the vertical part of the incision with alloy steel wire or Michel's clamps may be used for the latter if preferred. It is desirable to obtain an immediate union of the skin between the stoma and the drainage tube if no band of intact skin has been left. If a circular buttonhole incision has been used for the trachea in combination with either a vertical or a transverse incision the stoma may be completed before the larynx is removed.

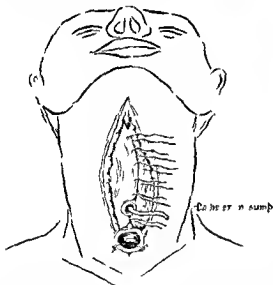


Fig. 417—Overlapping of ribbon muscles gives excellent support for pharyngeal closure. Sump drain of glass or alloy steel inserted under muscles (Steps 10 and 11). A small catheter is inserted into the sump and continuous suction maintained by a small electric pump for the first twenty four or forty eight hours sometimes longer.

Step 13 Dressing The incision is covered with a strip of boric-ointment gauze split in its lower portion to encircle the sump drain and the cannula. External to this several four by four inch gauze pads split vertically, are applied from below upward and from above downward beneath the neck plate of the cannula and surrounding the catheter which has been inserted into the sump drain. The dressing is secured by strips of waterproof adhesive and two-inch bandage. A no. 14 Fr. or no. 16 Fr. Levin feeding tube is passed through

the nose or if preferred a shorter tube (catheter) may be used (Fig. 418).

Postoperative Care—On return from the operating room, the patient is placed in a warm bed with the back rest raised to about 30 degrees. Room temperature is maintained to 70° F. and relative humidity should not be allowed



Fig. 418—Laryngectomized patient four days after operation. Note that skin clamps have been removed, as well as sump drain. The feeding tube is still worn in the nose.

to fall below 60 per cent. A double layer of moistened gauze laid over the cannula further assists in humidifying the inspired air. The tracheobronchial secretions are aspirated by means of a no. 14 Fr. rubber catheter introduced well below the end of the tracheal cannula. Aspiration is repeated every hour, or oftener if necessary, depending upon the amount and character of the secretion, and the inner cannula is removed and cleaned frequently by drawing through it, on a soft loop a strip of moist two inch gauze bandage. Aeration of the lower lobes of the lungs should be checked occasionally by auscultation.

Continuous suction is maintained through the small rubber catheter which has been inserted into the sump drain, if this should become obstructed, it can be removed and another sterile catheter inserted although this is rarely necessary. From 30 to 60 cc. of serosanguinous

drainage may be expected during the first twenty-four hours, the amount obtained during the second twenty-four-hour period is much less.

Sedatives are usually not required, if a sedative is needed, the coal tar analgesics, given by feeding tube, are preferred to opiates. Atropine should likewise be avoided, because of its drying effect on the tracheobronchial secretions.

Glucose water (5 per cent) may be given by nasal feeding tube in amounts of from 3 to 5 ounces every two hours, beginning about six hours after operation unless there is nausea. Tube feedings of strained fruit juice, milk, or eggnog may be started on the first postoperative day and increased gradually to provide adequate and well-balanced caloric intake. Additional amounts of cevitamic acid should be included in the feeding schedule. Perborate mouth wash should be used several times daily.

Penicillin intramuscularly, or one of the sulfonamide compounds by feeding tube, may be administered if appreciable wound contamination has occurred during operation. When a sulfonamide is used, it should be combined with sodium bicarbonate, and both fluid intake (at least 60 ounces per day) and urinary output should be accurately recorded.

The dressing is changed daily, inserting a sterile tracheal cannula. The entire sump drain is usually removed on the second postoperative day. Sutures or Michel's clamps may be removed from the vertical portion of the incision on the third, fourth, and fifth days; the sutures about the tracheal stoma should be left in place for a few days longer. Most patients are allowed out of bed on the third or fourth day (Fig. 418). The feeding tube may be removed as soon as the temperature has been normal for forty-eight hours, following a preliminary test-period of swallowing sterile water in small sips along side the tube, provided there is no evidence of wound infection or salivary leakage. Only liquids are given for the first few days after removal of the tube, and they should be taken slowly, thereafter pureed vegetables and other soft foods may be added to the diet, which is gradually amplified to include all kinds of solid foods.

Complications of Laryngectomy.—*Sepsis* is unquestionably the most common complication of laryngectomy, but it is much less common if the narrow-field type of operation is used, and

proper attention given to preoperative studies and treatment. As has already been stated, no patient with high blood sugar should be operated upon until every effort has been made to accomplish a maximum control, and oral sepsis, especially in the form of carious or abscessed teeth, should be entirely eliminated before operation. The current use of the sulfa drugs and penicillin both preoperatively and postoperatively will also lower the incidence of sepsis, and curtail its duration and lessen its severity when it does occur. In the presence of sepsis, it is of course necessary to see that adequate drainage is provided, in addition to administering whatever chemotherapeutic treatment may be decided upon. Irradiation lessens the vitality of tissues and greatly increases the danger of sloughing and suppuration, and therefore it is necessary to be especially vigilant in the care of wounds in patients who have had such treatment.

Hemorrhage should be a very rare postoperative complication in laryngectomy, but, if it does occur, the wound should generally be laid open and both pressure and sutures used to control it. One of the commonest predisposing causes of hemorrhage is sloughing associated with suppuration. Friable, sloughing tissues are, of course, difficult to ligate or suture. One primary indication, to the presence of hemorrhage, is to prevent blood from passing down into the tracheal stoma, and if blood does get down, aspiration and the removal of clots by means of a catheter or a bronchoscope is indicated.

Pharyngeal fistula is a not uncommon complication of laryngectomy, but it used to be much more common. The narrow field type of operation, with good overlapping of muscles and good conservation of blood supply, greatly reduces the tendency to this complication. Doubtless the good apposition of the edges of the pharyngeal wound, and the minimizing of infection from pharyngeal contamination accomplished by the use of the Brazilian clamp, will still further reduce its incidence.

Crusting of tracheal secretions (tracheitis sicca) gives some patients a great deal of trouble, and such crusts may become dangerously obstructive. They should be softened up by instillation of saline or glycerol, and then removed with the aspirator or, if necessary, with forceps. Prophylactically, the humidification of the air in the room and the wearing of

a moist pad over the tracheal stoma will help to prevent the formation of crusts.¹

Pulmonary complications after laryngectomy are rare. True pneumonia is very rare indeed, though bronchopneumonia and atelectasis may occur. These conditions, when they do occur, should be treated medically and by aspiration. Certainly the chest of the patient who has had a laryngectomy should be checked daily by the methods of physical diagnosis, and if there is any suspicion of a pulmonary complication, roentgen-ray examination should be done. The limitation of sedatives and particularly opiates, as well as drugs which dry the secretions (such as atropine), will reduce the incidence of pulmonary complications.

Hiccough occasionally occurs as an annoying but not serious complication. There is no one effective treatment, but sometimes the removal of the long feeding tube and the substitution of a short catheter will stop it. In other cases psychotherapy seems to be effective.

Toxic delirium is sometimes mentioned as a complication in elderly patients. It is not a common one and is rarely serious.

Irradiation Treatment—**TECHNIC**—Of course, individual patients are treated according to indications as they develop, but an outline of treatment for an average case is as follows:

One hundred and twenty five roentgens daily to each side of the neck for twenty five treatments, using a skin portal of 2 to 10 cm., and a skin target distance of 50 cm. The factors commonly used are 180 kv. (average) constant potential, 5 ma., and filtration with 2 mm. copper and 1 mm. of aluminum. Five to six roentgens per minute are given for about eleven and eight tenths minutes to each side. A total dosage of 3000 r or slightly more is delivered to each side.

In cases with metastatic nodes, protracted fractional irradiation should be supplemented by interstitial irradiation, using radon implants, preferably after neck dissection. When metastasis to the cervical nodes develops after operation, without local recurrence, neck dissection and radon implantation may be done without preliminary roentgen irradiation.

In the Department of Roentgenology and Radium Therapy of the Temple University Hospital the use of large rectangular fields was discontinued in 1936, in favor of open circular fields of 7.5 cm. and 10 cm. diameter, and during the last few years interchangeable circular metal cylinders (cones) 5.5, 6, 7, 8, 9, and 10 cm. in diameter have been employed. As stated

by Blady and Chamberlain,²¹ who have administered the irradiation in all of our cases, and whose work is freely quoted in this section, "with the smaller fields, the cutaneous reaction may be as severe as with the large fields, however, the area and volume of tissue involved are much less. This has made possible the administration of larger tumor doses without producing severe local and generalized constitutional reaction. With the smaller fields, severe reactions are more localized, better tolerated, and even at the height of a severe mucositis the patient is able to take soft and liquid nourishment with-



Fig. 419—The approximate center of the lesion is carefully localized and marked on the skin. A wire loop the size of the cone to be used is placed accurately over the outlined skin portal. This is fastened in place with adhesive tape and the patient is then placed in the exact position in which the treatment is to be given. A lateral roentgenogram is exposed and an accurate check of the selected center and the adequacy of the size of field can then be determined (Blady and Chamberlain²¹).

out the discomfort and pain that is experienced when the mucositis extends from the soft palate down to the base of the neck." However, as Blady and Chamberlain further state, "It must be emphasized that the use of cones requires accurate localization of the radiation beam. In all cases, the anatomic location of the lesion should be carefully centered on the skin of the neck after thorough localization of the lesion, by study with a laryngeal mirror, by direct laryngoscopy when indicated, by external palpation, and in the case of hypopharyngeal growths, by digital palpation of the lesion. The projected center and borders of the lesion are then marked



Fig. 420—A series of photographs showing the appearance of the skin during the course of treatment and after healing. A, Nineteenth day of treatment, total of 4750 r to each side of the neck. Treatment was completed on twentieth day at which time a total of 5000 r was delivered to each side of the neck, through a cone 5 cm. in diameter. B, Appearance of erythema four days after completion of treatment. C, Fifteen days after completion, there is definite blistering and central ulceration. D, Twenty-two days after completion, ulceration is severe although healing along the periphery of the field has taken place. E, Twenty-six days after completion, healing progressing. F, Fifty-seven days after completion of treatment, ulceration completely healed, although the newly-formed skin traumatizes easily as shown above. G, The appearance of the skin nineteen months after completion of treatment. (Blady and Chamberlain²¹)

on the skin, either with indelible dye or ink, or by tattooing with india ink." The center of the skin field should always be localized in relation to such fixed anatomic landmarks as angle of jaw, hyoid bone, superior cornu of thyroid cartilage, or cricothyroid membrane.

SKIN AND MUCOSAL REACTIONS—In the majority of cases, within a period of about five days after completion of treatment, there is a very marked superficial ulceration of the skin which, with proper surgical care, completely heals within a period of ten or fourteen days (Fig 420). It is important to care for the irradiation reaction as one would for any surgical case of severe burn. In the surgical care of these reactions, Blady and Chamberlain have found the following procedure very efficacious:

The raw surface is cleansed with dilute hydrogen peroxide and sterile distilled water, and then gently sponged with mineral oil. Gauze cut into desired shapes and sizes, either rectangular or circular, is heavily impregnated or overspread with an abundance of boric acid ointment. This sterile gauze is placed over the entire area of irradiation reaction. A dry dressing is fastened in place over this. These dressings are changed at least once or twice a day.

With this care the patients have been able to carry on their regular daily routines. Complete healing occurs in a period of ten to fourteen days, leaving a soft, pliable, and pale red skin. Gradually the redness blanches and the skin then begins to show varying degrees of pigmentation. Eventually the pigmentation disappears and telangiectasis appears.

"The mucosal reaction," according to Blady and Chamberlain,²¹ "usually appears after the skin reaction has become definite. If a dosage of 3,400 to 4,500 r is delivered to each skin portal, the acute mucosal reaction begins to appear. The early visible manifestation of reaction in the mucous membrane is a reddening which may be accompanied at times by lymphedema. Along with this the patient begins to complain of a 'scratching' sensation in the throat, or a 'sticking' or 'lumpy' sensation. Later, as the reaction becomes more intense, the act of swallowing is progressively more difficult. The formation of the whitish membrane or coating over the laryngeal mucous membrane is comparable to the ulcerative stage of irradiation reaction in the skin. It is interesting to note that even though the acute form of the mucositis appears later than the skin ulceration it is of shorter duration. Early in the

development of the mucous membrane reaction, an increased amount of mucus is formed. As the mucositis abates, the viscosity of the secretion is lessened and the symptoms of 'hawking' and spasmodic cough disappear. Treatment directed at cleansing the mouth and dissolving oral and hypopharyngeal secretions aids greatly in alleviating discomfort during this stage of the reaction. Irrigations carried out at intervals of from two to three hours with a solution of sodium bicarbonate and salt or alcaroid in lukewarm water are quite helpful."

COMPLICATIONS²¹—*Dyspnea* is seldom observed except in cases of bulky supraglottic tumors or subglottic infiltrative tumors, or advanced disease with infection. It is the opinion of everyone with experience in treating cancer of the larynx that if tracheotomy is likely to become necessary during the course of treatment by irradiation, it should be done before treatment is begun, and the irradiation not started until healing is complete. Our experience is no exception to this rule. Patients who have had to be tracheotomized during the treatment have shown a high mortality rate, while those who have had preliminary tracheotomy have survived.

Dysphagia is of limited importance in intrinsic laryngeal cancer, but when the cancer is in the region of the arytenoids, dysphagia may be quite pronounced, because the reaction is usually most intense at the tumor site. Lymphedema of the arytenoids, aryepiglottic folds, and pyriform sinuses is not infrequently observed, and when it occurs, there may be marked mechanical dysphagia. Anesthetic lozenges or powders will relieve soreness when it is due to reaction at the base of the tongue. When dysphagia interferes with nutrition, nasal feedings may be instituted. During the acute stage of the mucous membrane reaction, and especially in the presence of lymphedema of the arytenoids and the pyriform sinuses, or where bulky disease is present, utmost care should be observed when using a feeding tube if it is to remain in place for even several days. Sloughing of the hypopharyngeal or crico-esophageal mucous membrane may occur due to pressure necrosis. Nasal tube feeding formulas should be adequate in caloric content and should include vitamins and pureed vegetables.

Radiation necrosis is not frequently observed after administration of roentgen ray therapy alone, being more commonly encountered when sup-

plementary interstitial irradiation is used. In the presence of severe infections, however, radionecrosis may occur and, when the tumor has invaded the cartilage, the ensuing chondritis usually results in sloughing.

Hemorrhage may occur, either during the course of treatment or after its conclusion, as a result of necrosis. Of course, it can occur in the later stages, in patients who have not had irradiation. It is always difficult or impossible to arrest severe hemorrhage in the presence of sloughing. Tracheotomy can be done, and the larynx packed, in some cases. Vascular ligation is possible in others. Milder bleeding may be checked by cracked ice taken by mouth and ice applied externally.

Xerostomia may be produced when large fields, which include the submaxillary and parotid regions, are used, although in those patients receiving radiation through a small field it is not likely to occur. Oral irrigations with bicarbonate of soda and applications of gomenol to the mucous membranes at frequent intervals gradually relieve this condition. When the secretory function of the salivary glands has not been completely destroyed, it is possible to stimulate secretions by chewing gum or lemon peel, and by massaging over the glands.

Submental lymphedema is not infrequently observed, and, except for the resulting disfigurement, is of no consequence. Immediately after treatment lymphedema may be quite marked, it may continue intermittently and to a varying degree for months or possibly years.

Prognosis—Prognosis must be considered with respect both to life and to function, that is, in the case of the larynx, voice. Of course, *with out treatment*, cancer of the larynx is 100 per cent fatal, but the prognosis of the *treated* cases is better than in cancer of most other parts of the body.

Mortality—Let us consider first *operative mortality* and *mortality of irradiation*. The cases treated surgically should be divided into those treated by laryngofissure (or "partial laryngectomy"), and those treated by total laryngectomy. The mortality following the form of laryngofissure now used by the author should be practically zero. Of course, it cannot be said that any operative procedure is entirely free from risk, but it can be affirmed that in the author's clinic not a single death due to this operation has occurred during a period of fifteen years, and during this time between 100

and 200 laryngofissures have been performed. The mortality following total laryngectomy is certainly, according to the literature, much higher than that following laryngofissure. There have been some operative deaths even in the experience of our own clinic, but there has not been a single operative fatality in the last seventy laryngectomies done. Operative mortality in laryngectomy can certainly be reduced to a very low figure if certain precautions are observed. In the first place, there is no use in doing a laryngectomy on a patient who is a very poor operative risk, whether because of cardiovascular disease, pulmonary disease, or some other incurable complicating condition. The risk of serious infective complications is tremendously increased by the presence of an elevated blood sugar or the presence of oral sepsis. Also it must be admitted that the mortality is greater the more radical the operation must be. An other thing that greatly increases operative risk because of increasing the probability of sloughing and sepsis, is previous irradiation.

Treatment by irradiation results in some "treatment mortality." One of the most common causes of fatality during the course of irradiation, as noted previously, is *dyspnea requiring tracheotomy*. The same mortality does not attend tracheotomy if done prior to the institution of treatment. One of the greatest factors in reducing the morbidity and mortality of irradiation is the observation of the same preliminary precautions and preparation as in the case of patients who are to have laryngectomy (q v p 572).

There are those who insist upon the unique importance of the *end results* in the entire series of patients seen at a given clinic, as compared with the results obtained in any selected series. It has always been the author's contention that while the figures for the whole series of patients do possess a certain interest as vital statistics, the figures upon which our progress in the solution of the problem of selection and technic of treatment will be based, will be those obtained from the analysis of selected groups of cases, provided the basis for selection is clearly stated. A recent analysis of the end results in a series of 150 patients with cancer of the larynx treated at the Temple University Hospital by surgery and irradiation, during the period from 1930 to 1937, inclusive, showed a combined five year cure rate of 64 per cent. In fifty-one determinate cases in which laryngofissure was

done, an 80 per cent five-year cure rate was obtained, while in forty-two cases in which laryngectomy was done for an intrinsic lesion, in 69 per cent five-year cures were obtained. Therefore, we may say that of the 107 patients with *intrinsic* lesions who received surgical treatments by either laryngofissure or laryngectomy, eighty patients, or about 75 per cent, obtained at least a five-year cure. Nine patients with *extrinsic* lesions upon whom laryngectomy was done all died. During the same period, thirty-four patients were treated by irradiation (pro-

cords removed by *laryngofissure* practically always develop a useful voice, but its quality, pitch, and carrying power vary greatly. Cicatricial "adventitious" cords develop in most cases, and in the patients who develop good voices, generally either one good cord remains and a good "adventitious" cord has developed with which it can approximate and vibrate, or two good "adventitious" cords have formed to replace two cords that have been removed. Another mechanism by which a good voice can be produced is ventricular band phonation, which

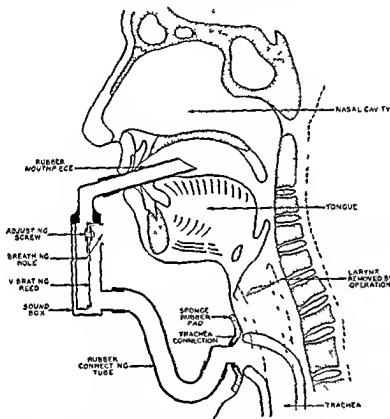


Fig. 421—Artificial larynx made by the Bell Telephone Company. Patients who do not wear a tracheal cannula use a pad such as is illustrated here. Those who do wear a cannula must attach the tubing to the cannula by a special connector.

tracted fractional technique), twenty-two of the cases intrinsic and twelve extrinsic, and in the *intrinsic* group a 59 per cent five-year cure rate was obtained, in the *extrinsic* group a 25 per cent five-year cure rate. Again let it be repeated, that the most significant and helpful figures are those obtained in clearly defined selected groups of cases by definitely stated and adequately explained methods of treatment.

Voice—Curing the cancer is of course the first consideration, but next comes voice. Patients who have had one or both true vocal

cords removed by *laryngofissure* practically always develop a useful voice, but which may afford an excellent and very efficient substitute for true cord phonation when one or both cords have been removed. The presence of a granuloma may interfere seriously with the production of a good tone, and therefore granulomas should be removed by direct laryngoscopy if they do not shrink up and disappear in a month or two after operation. Granulomas were noted in about 35 per cent of the cases in a recent series of fifty.⁷

The patient who has had a *total laryngectomy*, or is about to have one, is apt to be very depressed at the thought that he may never be able to talk again, but he will be cheered at once if he is told that the larynx is only the tone producer, and that the "molds of speech" (tongue, lips, cheeks, palate, etc.) which form the words are not disturbed when the larynx is removed. Theo it must be explained that he will simply need to learn to collect and control air in the back of his throat to replace the air column from the lungs, and to produce tone by means of whatever folds of the hypopharynx or pharynx may assume that function. He will probably be very skeptical about this, but he must be assured that experience has shown how to help patients get the knack of this sort of speech, and that his course of voice lessons will begin as soon after operation as healing permits. McCall²² even advises beginning the voice lessons before operation. The view expressed by Schall,²³ concerning not only speech but the social and economic rehabilitation of the laryngectomized patient, are fully supported by the replies received by the author²⁴ from thirty laryngectomized patients to whom a questionnaire was sent.

From his experience, the author believes it is a mistake for the patient to try the *artificial larynx* (Fig 421) before he has made an effort to develop a hucco-esophageal voice without apparatus. This apparatus should, in the author's opinion, be reserved for those who are not able, after a fair trial, to develop the more natural kind of speech.

Following *irradiation* the vocal results are better than they are after any surgical procedure. In fact, the voice may be expected to return completely to normal, or almost normal, but this must not lead us to choose irradiation if the ultimate prognosis as to cure of the disease and preservation of life is not so good, unless operation is for some reason contraindicated, or is refused by the patient. In the experience of Blady and Chamberlain,²¹ "Where the disease is extensive, and destruction of the cords has occurred prior to the treatment, definite impairment of voice is an unavoidable sequela." They have noted also, that when a second series of treatments is required, very marked impairment of voice generally occurs.

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BENIGN TUMORS OF THE LARYNX

A benign tumor of the larynx¹ is, strictly speaking, a true neoplasm of benign character arising from the larynx. As a matter of fact, the majority of "benign tumors" of the larynx are not true neoplasms. The term "benign tumor" has been quite generally applied to lesions tumor like in appearance and symptoms, which are really of inflammatory origin and which do not show histologically the characteristics of a neoplastic process. The terms "fibroma," "angioma," and "fibroangioma" are often applied incorrectly to such lesions, which are really retention cysts, hematomas, or polyps, but not true neoplasms.

Etiology.—In the case of papilloma, for example, irritation and infection are both considered to be possible causative factors. In the non neoplastic benign tumors, inflammation may be the important etiologic factor, or trauma. Trauma may be of recurring character, as in the case of persons who habitually abuse the larynx, or it may be the trauma of a single injury such as that due to shouting or straining to produce a forceful note in singing.

Pathology.—As was stated by New and Erich,² "benign tumors are composed of well differentiated cells which displace rather than infiltrate tissues. They never metastasize." Of course, the differentiation between benign and

malignant tumors is extremely important from the clinical point of view, because the treatment is so different. The differential diagnosis should always be made on a histopathologic basis rather than on gross appearances. Biopsy is indicated in all cases of tumors or tumor-like lesions of the larynx. The differentiation between true benign tumors and benign tumors of non neoplastic character is less important from the practical viewpoint. The gross and microscopic pathologic characteristics of various kinds of benign tumors will be considered under appropriate headings in the succeeding paragraphs.

Symptoms.—*Hoarseness* is by far the commonest symptom of benign tumors of the larynx, and in fact, this symptom eventually develops in almost every case, whether or not the lesion is located on the true vocal cord. *Dyspnea* will be produced if the tumor becomes large enough to cause obstruction. It develops earlier in lesions of the subglottic region. A pedunculated tumor may cause intermittent hoarseness and dyspnea, depending on change of position. *Cough* may occur, but is not a common symptom, and this is true also of *hemoptysis*. There may be a subjective sensation of "lump in the throat," but this is noted only in association with the larger tumors, and generally those of the extrinsic area. *Pain* and *dysphagia* are occasionally complained of, in cases in which there is complicating ulceration and infection.

Diagnosis.—The diagnosis of benign tumors of the larynx is made by inspection with the mirror or with the direct laryngoscope, supplemented by histologic examination of tissue removed for biopsy study. In the case of adults, the larynx can generally be well seen by means of a laryngeal mirror, but for the removal of tissue for examination direct laryngoscopy is preferred by the author. In children direct laryngoscopy affords the only practical means of examination of the larynx. Of course, in addition to examination of the larynx and histopathologic study of tissue removed, there should be a careful history, a general physical examination, and studies to exclude tuberculosis and syphilis, such as roentgen-ray examinations of the chest and blood serum tests. Roentgen-ray examination is useful, not only to exclude tuberculosis by demonstrating normal lungs, but also, especially in the larger tumors, to demonstrate the position, shape, and location

of the growth by roentgenography of the neck in both lateral and anteroposterior projections. The development of planigraphy has marked a very great advance in the possibilities of roentgen ray study in this region.

Complications—*Asphyxia* is perhaps the most frequent complication of benign tumor, but it does not occur except when the tumor or (in the case of multiple papillomas) tumors have become of sufficient size to obstruct the glottis. This complication must be treated by immediate removal or by tracheotomy. *Myasthenia laryngis* may also develop as a compli-

the site of a malignant one. Removal can be accomplished by the use of curved forceps under the guidance of the laryngeal mirror, but the author's preference is for removal by direct laryngoscopy in most instances (Figs 422-423). In the case of very large tumors, suspension laryngoscopy under general anesthesia affords better exposure and is perhaps preferable. For the great majority of benign tumors, however, the use of the suspension method is quite unnecessary, since simple direct laryngoscopy under local anesthesia is perfectly satisfactory.

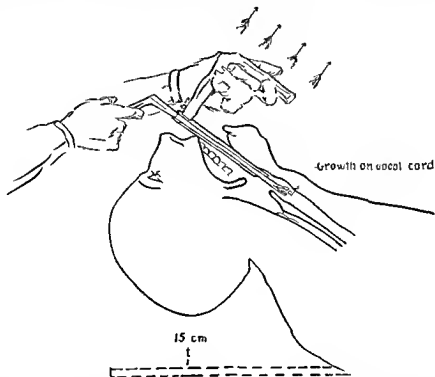


Fig. 422—Direct laryngoscopy for the removal of a benign tumor of the vocal cord. For this operation the smooth-tipped anterior commissure laryngoscope and the angular cupped forceps are the best instruments in most cases (see Fig. 423). Hands are sketched ungloved to show exact finger placement.

cation of benign tumor from prolonged phonation against the obstruction of the tumor mass which prevents perfect approximation of the cords. Some degree of myasthenia may remain as a sequela of benign tumor, even after it has been removed. Unfortunately another not uncommon sequela is permanent impairment of voice by unskilful instrumentation at removal.

Treatment—Benign tumors should be removed in practically every case. Removal is indicated because of impairment of voice or obstruction of the airway and because of the possibility that the benign tumor may become

Prognosis—The prognosis of benign tumors of the larynx is excellent insofar as life is concerned because these lesions never prove fatal except if allowed to become large enough to obstruct the airway. With regard to voice the prognosis may also be considered excellent, because after removal of the tumor if carefully done the voice usually returns to normal or practically normal. Benign tumors rarely recur with the exception of papillomas.

Types—*Papilloma*—A papilloma is a benign epithelial tumor of papillary structure having a core of vascular connective tissue. It is the commonest of all benign tumors of the

larynx, occurring in individuals of all ages. Papillomas may rise in any part of the larynx, but they do not invade the submucosa. They have a marked tendency to recur after removal, but only very rarely do they undergo malignant change. The diagnosis of papilloma is made tentatively by inspection with the mirror or the direct laryngoscope, but in all cases the diagnosis should be confirmed by biopsy.

The best treatment, in the author's experience, is simple scalping off by means of direct laryngoscopy. Great care should, of course, be taken not to damage the underlying normal tissues. In cases of papillomas showing unusually rapid tendency to recurrence and histo-

in children, because of subglottic extension of the tumor and of too rapid recurrence to permit the adequate removal of the papillomas. Certainly tracheotomy properly done, with no treatment directly to the papillomas themselves, is preferable to radical surgery or radical irradiation, in view of the ultimate self-limitation of the disease.

Adenoma—This is a rare tumor in the larynx. When it does occur it is not infrequently cystic. The diagnosis is made by direct laryngoscopic biopsy and the treatment is simple avulsion by direct laryngoscopy.

Fibroma—True fibromas are very rare tumors of the larynx, but the term "fibroma" is very

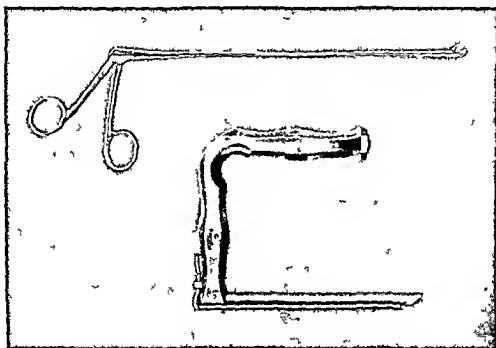


Fig. 423—Direct laryngoscope of smooth tipped anterior commissure type and angular cupped forceps

pathologically the characteristics of rapid growth, deep roentgen ray therapy may be used. However, if it is to be used at all, irradiation should be given in a dosage almost as great as that which would be used for carcinoma. Smaller doses will not be effectual, and may even stimulate growth. In treating papillomas it must always be borne in mind that the disease is self-limited. In children, even in those whose whole larynx is filled with papillomas, and though the growths recur quickly after removal, self limitation is generally observed at puberty or a little before. Adults who develop papilloma have generally not had papillomas in childhood. Tracheotomy is quite often necessary

often applied to benign tumors of inflammatory or traumatic origin, as for example, organizing hematomas (q1) or fibrous polyps. The diagnosis of true fibroma is made by examination and biopsy. Treatment is, of course, surgical removal. In the case of smaller tumors, this can be done by direct laryngoscopy, but in the large ones suspension laryngoscopy or a surgical procedure such as thyrotomy or pharyngotomy may be necessary. Tracheotomy is generally required as a preliminary procedure in such cases.

Neurofibroma—Neurofibroma is a rather rare tumor in the larynx. These tumors arise from the cells composing the sheath of Schwann

which are of ectodermal origin. A typical case seen at the author's clinic has been reported by Van Loon.⁴ The *diagnosis* of neurofibroma is made by laryngoscopic examination and roentgen study, including planigraphy (Fig. 424). *Treatment* of neurofibroma is removal, by endoscopic procedures if possible, and if not, by thyrotomy, as in Van Loon's case.

Angioma—Angioma is a term very commonly applied incorrectly to organizing hematoma, because of the presence of blood cells and blood vessels. The terms "angioma" and "fibro angioma" should however, be reserved for true neoplasms which arise from blood or lymph vessels. Angiomas may be either hem-

atoma as a preliminary procedure, though this may not be necessary in the case of small tumors. While the author believes that an attempt should always be made to distinguish between true angiomas and the "pseudo angiomas" which are of inflammatory or traumatic origin, sometimes this differentiation is difficult to make on a histopathologic basis alone.

Myxoma—Tumors of muscle tissue occur in the larynx but very rarely. Their diagnosis and treatment is similar to that of other benign tumors.

Myxoma—This tumor occurs not uncommonly in the larynx though it is probable that

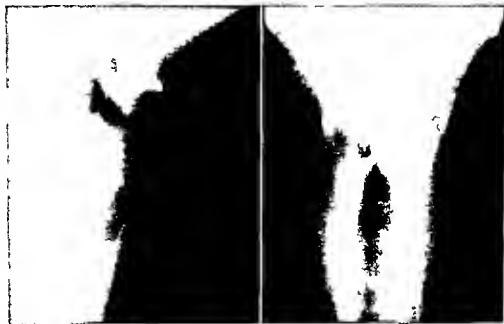


Fig. 424—Neurofibroma of left side of larynx, especially well demonstrated by planigraphy. This tumor was removed by thyrotomy with an excellent result (Van Loon⁴).

angiomas or lymphangiomas, but the former are by far the most common. Hemangiomas may be either simple or cavernous. They sometimes occur in infants who have hemangiomas on other parts of the body. Like most other true benign neoplasms of the larynx, true angioma is rare. When it does occur, the *diagnosis* is made by examination and biopsy. A typical case of angioma of the larynx is reported with an illustration of laryngoscopic appearance, by Jackson and Jackson.¹

Treatment of angiomas is generally surgical removal of the tumor by direct laryngoscopy, but due precautions must be taken to control hemorrhage. Generally it is best to do a trache-

otomy as a preliminary procedure, though this may not be necessary in the case of small tumors. While the author believes that an attempt should always be made to distinguish between true angiomas and the "pseudo angiomas" which are of inflammatory or traumatic origin, sometimes this differentiation is difficult to make on a histopathologic basis alone.

many of the myxomatous tumors reported are really of inflammatory origin. Myxoma are composed of loose connective tissue containing branched stellate cells in a matrix of viscid mucoid material. If fibrous tissue predominates, the tumor is called a fibromyxoma. Any connective tissue tumor may undergo metaplasia and become myxomatous. Clinically, these tumors cannot be distinguished from inflammatory polypoid growths. *Treatment* is the same as for tumors of inflammatory origin, or any other benign laryngeal tumors of similar size and location.

Chondroma—This tumor, though not common in the larynx, occurs frequently enough to

be quite important Chondromas generally arise from the thyroid cartilage or the cricoid cartilage Not infrequently ossification occurs in them, and if there is a considerable amount of ossification they are called "osteochondromas" Symptoms of chondroma are dyspnea and dysphagia Hoarseness is generally noted also, though it is a less important symptom in chondroma than in most other benign tumors of the larynx Because of the slow growth of these tumors, the symptoms are generally insidious in their development Dyspnea may very gradually increase in severity, and then suddenly become so severe that the patient may asphyxiate if emergency tracheotomy is not

required, and occasionally total laryngectomy will be necessary

Benign Tumors Not Strictly Neoplastic—In the clinical sense any mass of abnormal tissue is a "tumor," even though not pathologically a neoplasm

HEMATOMA (ORGANIZING HEMATOMA)—This is probably the commonest kind of non neoplastic tumor of the larynx Hematomas develop as a result of voice strain, as in shouting or forceful speaking or singing. Their pathologic development is that of hematoma anywhere, and after a time they undergo organization (Fig 425) In their early stages they may simulate angioma, in their later stages fibro



Fig 425—Histopathologic structure of benign tumors of inflammatory origin which may be called organizing hematomas They are not true neoplasms

performed The diagnosis may be made by palpation in the case of the tumors that develop on the outside of the thyroid or cricoid cartilages If the tumor develops on the inside it, will be visible on indirect and direct laryngoscopy as a smooth, hard bulge, generally covered with vascular network On palpation with the fingers externally, or a probe internally, the mass will be found quite hard Obtaining a biopsy specimen is generally difficult but should be attempted The roentgen ray examination of the neck, including planigraphy, is of considerable value Treatment depends upon the size and location of the tumor, but generally some form of partial laryngectomy is

angioma or fibroma Clinically hematoma appears as a rounded reddish mass of tissue on the cordal margin which interferes with phonation (Fig 426) Treatment is by direct laryngoscopic removal, or in some instances, removal under the guidance of the laryngeal mirror In either case, great care should be taken to scalp the mass off superficially without damaging the underlying normal tissue of the cordal margin

POLYP—This is another common form of benign tumor that is not a true neoplasm Polyps may vary greatly in size and shape Some are sessile (polypoid corditis) and others are pedunculated These polypoid tumors are

similar to myxomas grossly and histologically *Treatment* is simple removal by direct laryngoscopy

VOCAL NODULES—These are typically bilateral, though not necessarily so. They appear as small nodules on the cordal margins, at the junction of anterior and middle thirds (Fig

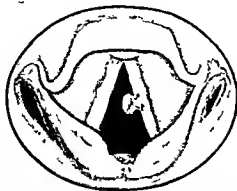


Fig 426—Reflected (indirect) view of organizing hematoma of the larynx. This benign tumor of inflammatory origin is often misnamed angioma or fibroma.

427). They are sometimes called "singer's nodes" or "screamer's nodes." They are also sometimes called "fibrous nodules." They are said to be due to singing or speaking out of normal vocal register, or attempting to produce volume of greater than normal capacity. In the author's experience, a few of the patients have been singers, but more have been school teachers. Histopathologically these nodules vary, some showing fibrous inflammatory tissue and some loose vascular tissue. They are generally covered with thickened epithelium, and the stroma consists of fibrous connective tissue, leukocytes, and more or less organized inflammatory tissue (Fig 428). Vocal nodules produce a voice change which is not exactly a hoarseness, it is rather an uncertainty of voice tone. Of course, if the nodules become enlarged, they may produce hoarseness.

Treatment has been the subject of a considerable amount of discussion. It has been urged by some that vocal nodules should not be removed, that treatment should consist in vocal rest and modification of the patient's method of speaking or singing. While there can be no doubt that both of these measures are of value and may be curative in some cases, it is the author's opinion³ that in most cases the careful removal of the nodules under direct laryngoscopy, using a special cupped forceps, is

indicated. However, if the nodule is reddened and inflamed, a preliminary period of vocal rest should be advised, especially in the case of a professional singer. Vocal nodules have been removed by the indirect method, but so great is the need for accuracy in order to remove the nodule cleanly without damaging the cordal margin that the direct method is definitely to be preferred. The fixation of the cord obtainable by the direct method contributes greatly to the accuracy of removal. In the cooperative adult patient, local anesthesia is perfectly satisfactory, but in a child or apprehensive adult, the use of a general anesthetic, as for example intravenous pentothal sodium, is justifiable. Immediate improvement in voice is to be expected following removal of nodules.

CYSTS—Most cysts occurring in the larynx are non-neoplastic. The commonest form is the mucous cyst, formed because of obstruction of a duct, and accumulation of secretion in a mucous gland. There are, of course, occasional embryonal cysts, which are congenital. The most frequent site of cysts in the larynx is the anterior surface of the epiglottis, cysts originating in this region may fill the valleculae. The next most common site is the vocal cord. Small cysts of the cord margin not infrequently simulate vocal nodules, though on close inspection they can generally be differentiated by their

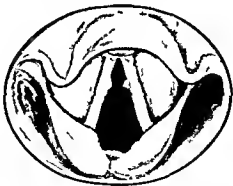


Fig 427—Reflected (indirect) laryngoscopic appearance of pair of vocal nodules.

opalescent appearance. *Treatment* of cysts of the larynx consists in superficial removal preferably by direct laryngoscopy.

EVERSION OF THE VENTRICLE—This may simulate a cyst. It is, as the name implies, simply an out-pouching of the ventricle of Morgagni into the laryngeal lumen. Sometimes the sacculus is also everted. While simple eversion does

occur, more often there is some other associated morbid condition. The principal *symptom* produced by eversion is the impairment of voice produced by the damper-like action of the everted ventricle upon the vocal cord. Eversion may be unilateral or bilateral. *Diagnosis* is made by direct laryngoscopy. By this means eversion may be differentiated from other kinds of tumors, cysts, polypoid corditis, and other conditions. The *treatment* indicated in most cases is removal of the everted tissue with a sharp cutting forceps. In some cases of eversion, however, the tissue can be tucked back in place with a closed forceps and it will remain there, at least for a time.

Diagnosis is made by examination of the larynx and the neck, and by roentgen ray study. The advent of planigraphy has brought us a peculiarly efficacious roentgen ray technic of diagnosis of this condition (Fig. 430). *Treatment* depends on the size and type of sac. Very often the "internal" laryngoceles do not require treatment. The external ones should be excised (Fig. 431).

KERATOSIS—This condition is not really a tumor. It may be defined as "a localized cornification of epithelium without invasion of the subepithelial tissue" (Jackson and Jackson¹). Pathologically the condition is characterized by piling up of epithelium which undergoes



Fig. 428—Histopathologic structure of vocal nodule removed from the cord of an operatic singer

LARYNGOCELE—This is a tumor-like lesion consisting of an anomalous air sac communicating with the laryngeal ventricle. Coughing may enlarge the sac, but it is probably always a congenital condition, an evolutionary remnant of the huge air sacs seen in the anthropoid apes. In typical cases the sac communicates with the *sacculus*, and it may herniate through the thyrohyoid membrane and become visible on the outside of the neck between the hyoid bone and the thyroid cartilage as a soft elastic swelling that bulges on coughing (Fig. 429). The laryngeal appearance varies, but in most cases the sac is visible, protruding from the ventricle, or distending the ventricular band. *Symptoms* are impairment of voice, and occasionally dyspnea.

cornification. There may be hyperkeratosis of the epithelium of one or both cords, or the proliferating epithelium may take the form of small, whitish pointed projections springing directly from the mucosa of any part of the larynx. There is usually an associated chronic inflammatory process diffused over the mucosa, and sometimes a small, reddish areola surrounding the horny projection. Hoarseness is the characteristic symptom. *Diagnosis* must be made by biopsy, though the gross appearance is fairly characteristic. *Treatment* is removal of the whitish projections by direct laryngoscopy. Growths may recur but malignant changes are unlikely.

LEUKOPLAKIA—This is seen as a whitish

patch or patches on the mucous membrane. It is suspected of being "precancerous," though it is not necessarily so in every case. The epithelium is markedly thickened and shows

Granuloma.—This may be *specific*, that is, a manifestation of specific systemic infections such as syphilis or tuberculosis, or *nonspecific*. The specific granulomas will be discussed

Fig. 429.



Fig. 430.

Fig. 429.—A bulge on the right side of the neck produced by an external laryngocoele (see Figs. 430, 431).

Fig. 430.—Laryngocoele shown in (left) ordinary anteroposterior roentgenogram and (right) planigraphic anteroposterior film. Note that in this section the communication between the external and the internal portion of the laryngocoele is well shown. This sac was removed by external incision. (See Figs. 429 and 431.)

hyperkeratosis. Epithelial pegs are exaggerated. *Treatment* is excision whenever possible, and if this cannot easily be done, close observation should certainly be advised.

elsewhere. Nonspecific granuloma is most commonly superimposed on a contact ulcer (*q.v.*).

Amyloid Tumors.—These are uncommon, but

when they do occur, diagnosis can be made by biopsy (Fig 432) Treatment will depend on the site and extent of the amyloid deposits, but

and five lesions of the diffuse subepithelial infiltrative type

Xanthoma—Xanthomatous tumors are rarely seen in the larynx, though they are relatively common in the skin Grossly, these lesions appear as smooth nodular areas or plaques, yellowish in color A typical case is illustrated elsewhere¹ Histologically they show cells of various sizes in a stroma of connective tissue The most characteristic cells are the large "foam cells" containing lipoid deposits Analysis will generally show an increase in the concentration of cholesterol and lipoids in the plasma The condition may accompany diabetes, nephritis, jaundice, or pregnancy Treatment is unsatisfactory The lesions may be excised or coagulated, but new lesions are likely to develop Occasionally they disappear without treatment Irradiation is of no value Xanthomatous lesions have been known to cause obstruction through size, or through contracture in the 'regressive' phase, and, of course, this must be relieved, by tracheotomy or otherwise

Thyroid Tumors—Aberrant thyroid tissue may appear in the larynx as a tumor-like mass Not infrequently it will be found to show malignant developments but it may be benign

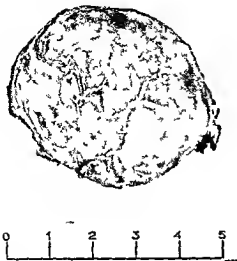


Fig 431—External laryngocele removed by external operation Compare Figures 429 and 430

excision is to be desired if it is possible without too radical resection Since the condition is generally almost static, radical treatment is



Fig 432—Section showing amyloid deposits in a case of laryngeal amyloidosis This tissue was removed for biopsy by direct laryngoscopy The patient had marked thickening of the ventricular bands At one time the diagnosis of dysphonia plicae ventricularis had been made

contraindicated New and Erich² report eighteen cases, four localized tumors, nine tumors which had undergone amyloid degeneration,

Diagnosis is by biopsy and treatment by excision, or, in some cases, irradiation

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PACHYDERMIA OF THE LARYNX

Pachydermia of the larynx is a localized epidermoid thickening of the laryngeal mucosa. Virchow, who discovered it as a pathologic entity, called it a 'wartlike pachydermia' (*pachydermia verrucosa laryngis*).

The disease is really uncommon. Most of the cases in the literature were errors of diagnosis based on examination with the mirror without biopsy.

Etiology.—The cases described by Virchow occurred in nocturnal carousing drunkards. This combination of shouting, roaring, and singing out of doors with chronic alcoholism seems to be the chief causative factor in the cases to which the term can be properly applied.

Pathology.—Grossly, Virchow described a tumor-like formation, without ulceration, in the posterior commissure and extending forward to cover both sides of the glottis posteriorly. In a few cases the masses of epidermoid thickening have extended over other parts of the larynx and downward to the trachea, with a sharp border line of demarcation at the junction with the normal. The grayish epidermoid tissue, as described by Virchow, could be dragged off in strips. Examined microscopically the strips were found to be thickened epithelium undergoing epidermoid changes. The thickened cells in some of our cases² were vacuolated. There was much irregularity in the outline and position of the basal-cell layer. Pegs extended upward from the connective tissue into the thickened epidermoid tissue, but at no place was there a breaking through of the epidermoid cells into the connective tissue. Two pathologic characteristics are especially to be noted: there is no ulceration and the border adjacent to normal mucosa is definitely demarcated.

Laryngeal Appearances.—Examination with the laryngeal mirror reveals a mass of grayish tissue in the posterior commissure extending forward on both sides, usually symmetric in form, but in a few cases unilateral. The mass in some cases is nodular. On phonation the mass forms two masses, one on each side, which come together and hold the cords from perfect approximation. On inspiration strings of viscid secretion draw out and bridge across from one side to the other.

On direct examination the mass of tissue is found on palpation with closed forceps to be firm and, when properly presented, to be free of ulceration.

Symptoms.—Hoarseness and a rough deep-toned voice are usually conspicuous symptoms in cases of a large growth. Soreness and a sense of fatigue after prolonged use of the voice and after much coughing effort to get out sticky secretion are complained of in some cases. Dyspnea is present when the growths are large.

Diagnosis.—The location and form of the growth and its freedom from ulceration are characteristic. Tuberculosis, granuloma, and cancer all occur in this region. The only certain method of diagnosis is by biopsy. Microscopic appearances of tuberculosis, granuloma, and cancer, as described elsewhere herein, are characteristic, and in pachydermia no ulceration is found, whereas in contact ulcer there is no pachydermia.

Treatment.—Operative removal of epidermoid tissue is clearly indicated. As described in connection with removal of benign growths, utmost care and precision are necessary to avoid injury to normal tissues. Occasional cases of extension over a wide area require extensive scalping off of the epidermoid tissue, but with proper care in the use of the cupped forceps no harm need be done. The growths do not ordinarily recur after precise removal, and perfect function is usually restored.

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HYPOPHARYNGEAL DIVERTICULUM

Pouches opening into the esophagus or pharynx are found in dogs, horses, pigs, ant-eaters, sloths, bats, and other animals. Related sacs found in the monkey have a physiologic function in the production of sound. In men congenital pouches are very rare but when present may extend into the pyriform sinus and cause suffocative attacks during swallowing. An acquired form that is relatively common develops chiefly in men over fifty years of age as a hernia like protrusion of the posterior wall of the hypopharynx through the diverging fibers of the cricopharyngeal muscle. Favored by the muscular weakness of age and perhaps excited by the gulping of large morsels of food, the weakened wall stretches into a pulsion diverticulum. Chevalier Jackson points out that the herniation may result from the failure of the cricopharyngeus to relax ahead of the bolus of food that is being pressed down into the esophagus, or, as he describes it, 'failure of coordinate opening of the cricopharyngeal pinch cock'. The pouch, on repeated distention with liquids and food, tends to enlarge progressively and to emerge at the left side of the esophagus, sometimes producing a palpable or visible swelling in the neck. Under traction the opening to the diverticulum gradually replaces in situation and size the normal opening into the esophagus, the latter then being represented by a small, puckered opening displaced to the anterior wall of the hypopharynx, which may be difficult to find with the esophagoscope. The pulsion type is much more common than the small traction diverticulum which occasionally is found in the thoracic esophagus, commonly due to adherent tuberculous lymph nodes, and rarely producing symptoms or requiring treatment. As the pulsion sac enlarges it may enter the thorax and anterior mediastinum and acquire a capacity of 500 cc. or more. Food may be retained in the sac, and decompose with resulting irritation or, rarely, ulceration, perforation, or malignant change.

Symptoms.—Patients with the pulsion type of hypopharyngeal diverticulum experience a progressively increasing dysphagia, with regurgitation of food, eructation of gas which may be fetid, and a sense of pressure and distress in the neck. Over a third of patients complain of persistent cough and excessive secretion of mucus, many of choking or strangling. Nearly one

third mention embarrassing gurgling noises referred to the left side of the neck, which also may be produced on swallowing or by pressure over the diverticulum. The patients examined at the Jackson Clinic have been from thirty to eighty nine years of age, the majority being between thirty and seventy years. Seventy per cent were males having a history of symptoms of from one week to fifteen years' duration. In about 50 per cent of the patients the sac was large, in 30 per cent small. From pressure on the recurrent nerve and cervical sympathetic

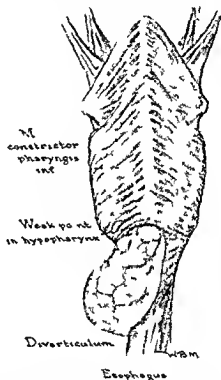


Fig. 433.—The pulsion diverticulum of the esophagus or more properly the hypopharynx is a herniation largely of pharyngeal mucous membrane through the cricopharyngeus muscle or the weak lozenge shaped area above it. Originating posteriorly, the sac usually passes down the left of the esophagus.

fibers, especially when inflammatory or malignant change has occurred, hoarseness and Horner's syndrome may be produced. Many of the patients lose weight and a few have developed pneumonitis or pulmonary abscess from aspiration of contents from the sac. Finally the esophagus may be so obstructed even without inflammatory or malignant change, that starvation results.

Diagnosis.—The diagnosis is best made by anteroposterior and lateral roentgenograms taken after the swallowing of a barium mixture,

which will reveal the size and position of the diverticulum. A bougie or esophagoscope should not be used blindly as it may perforate the bottom of the thin-walled sac. However, with the films as a guide, an esophagoscope may be passed, the sac emptied, and the wall examined. The esophagoscopic appearances in a pharyngeal diverticulum are described by Jackson and Jackson¹ as follows:

"The esophagoscope will without difficulty enter the mouth of the sac which is really the whole bottom of the pharynx, and will be arrested by the blind end of the pouch, the depth of which may be from 4 to 10 cm. In some cases the bottom of the pouch is in the medias-

till the occipital half of the head is below level of the table. Between the orifice of the sac and the orifice of the subdiverticular esophagus are some of the fibers of the cricopharyngeus muscle which close the subdiverticular orifice, whereas the orifice of the sac is patulous."

Treatment.—Treatment consists in the operative removal of the sac and a water-tight closure of the remaining opening without altering the normal caliber of the esophagus. This requires an external incision. Various methods for the division of the partition between the pouch and esophagus, the inversion, transplantation, or rotation of the sac without removal have been

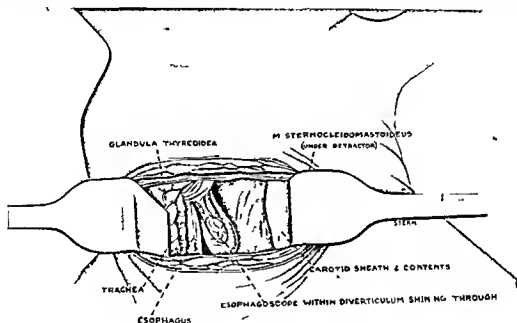


Fig. 434.—Aided by an esophagoscope introduced from the mouth into the sac, the diverticulum is readily exposed through a short transverse incision at the level of the cricoid cartilage. The exposure is nearly bloodless and it is not necessary to divide any important structure. The thyroid lobe is retracted medially and the sternocleidomastoid muscle and carotid sheath laterally, while the omohyoid muscle lies medial and below the operative field.

tum. The walls are often pasty, and may be eroded, or ulcerated, and they may show vessels or calcines. On withdrawing the tube and searching the anterior wall, the subdiverticular slitlike opening of the esophagus will be found though perhaps not always easily. The esophageal speculum will be found particularly useful in exposing the subdiverticular orifice and through this a small esophagoscope may be passed into the esophagus, thus completing the diagnosis. Care must be exercised not to perforate the bottom of the diverticular pouch by pressure with the esophagoscope or esophageal speculum. The walls of the sac are surprisingly thin. In passing the tube into the sac the head of the patient should be high in the correct position. To find the subdiverticular orifice, however, the anterior wall must be searched, and for this the head must be dropped

found unsafe or unsatisfactory and should not be used. The earlier crude excision with ligation of the neck of the sac was followed by such frequent leakage and secondary and often fatal mediastinitis that surgeons turned to a two- or three stage operation. In the first stage the sac is exteriorized, elevated, perhaps rotated, and anchored near the skin. Ten to fourteen days later, protective adhesions having formed, the sac is excised and the wound permitted to close by granulation or with the aid of sutures. Between stages the sac occasionally becomes gas-distended or gangrenous with sufficient pressure

upoo the trachea to cause dyspnea and to require prompt evacuation or excision. In the removal of the adherent sac it is difficult to locate the line of attachment to the hypopharynx. If too much tissue is removed as is not uncommon the use of dilating bougies may be required for the ensuing year, or, if the sac is not completely excised a residual pouch may give

trational operation, secondary abscess in the neck (9 per cent), incomplete removal of the sac (36 per cent), and secondary stricture requiring prolonged esophageal dilation (9 per cent).

With improvement in technic the one stage operation has become very safe and the sac is now so accurately removed that secondary bouginage is not required, the period of disabil-



Fig 435

Fig 435—From the light in the end of the Jackson esophagoscope the diverticulum gives off a reddish glow in the depths of the wound enabling it to be immediately recognized and grasped by nontraumatizing visceral forceps.

Fig 436—The diverticulum having been isolated from the surrounding tissues the esophagoscope after emptying the sac by aspiration is withdrawn and guided by a previously swallowed thread into the esophagus proper. The neck of the sac is divided and the slit like opening in the hypopharynx very accurately closed by a continuous inverting suture. Meanwhile the esophagoscope removes esophageal and pharyngeal secretions and serves as a mandrin preventing undue constriction of the esophagus in suturing so that the later passage of bougies is unnecessary. In this drawing part of the esophageal wall is omitted to show the esophagoscope and string.

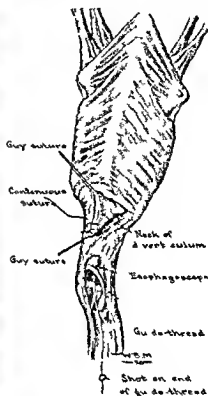


Fig 436

later trouble. In any case convalescence is prolonged by the presence of an open, granulating wound, and a disfiguring scar is left upon the neck, and, while rare death has occurred from mediastinitis following the two stage operation. The following complications have been reported from the stage operation: tracheal or esophageal obstruction between stages from the sac filling with air or becoming gangrenous, recurrent laryngeal nerve paralysis (9 per cent), persistent esophageal fistula requiring addi-

tion is much less than with the multiple-stage operation, the patient often being able to leave the hospital, completely healed and with an almost invisible scar, by the seventh or eighth day, while the danger and discomfort of a second or third operation and anesthesia are eliminated. In our series of cases, in association with the Chevalier Jackson Bronchoscopic Clinic, there has been no death from mediastinitis or infection and no persistent fistula or sinus.

Exceptionally the patient is found to be so

starved and dehydrated that parenteral feeding is required to prepare him for the operation. The operation is nearly bloodless and free from shock, and feeding through an indwelling nasal tube may be started immediately afterward.

Technic of Operative Removal of Diverticulum.—Following the plan of esophagoscopy assistance devised by Chevalier Jackson, one or two days before the operation the patient swallows a strong silk thread with two or three bird shots clamped near the lower end. After this has passed some distance into the intestine the thread is not easily withdrawn, and when threaded through the esophagoscope serves as a valuable guide in quickly locating the subdiverticular orifice of the esophagus. Meanwhile, attention is paid to the hygiene of the mouth and nasopharynx without, however, producing inflammatory reaction. The patient comes to the operating room moderately narcotized by a preliminary subcutaneous injection of morphine sulfate (0.01 gm.) and hyoscine hydrobromide (0.0004 gm.). The nasopharynx is sprayed with a solution of cocaine or butyn. The skin of the anterior neck is infiltrated at the level of the cricoid cartilage with a 1 per cent solution of procaine to which 1 minim of epinephrine is added for every 10 cc., provided there is no contraindication. A 6-cm. needle is then entered over the sixth spinous process on each side and about 45 cc. of the solution injected over but not into the three adjacent interspinous foramina. Sufficient of a 1 or 2.5 per cent solution of pentothal sodium is injected intravenously to produce relaxation while the esophagoscope is in use. For an inconspicuous scar a transverse incision is made at the level of the cricoid cartilage, extending from the midline of the neck to the left for about 7 to 8 cm. After dividing the skin and platysma, the deep layers are separated through the bloodless space lying between the larynx, thyroid recurrent nerve, and esophagus medially and the left sternomastoid and carotid sheath laterally until the fascia covering the cervical spine is reached. With proper retraction it is unnecessary to divide the omohyoid muscle or superior thyroid vessels. The diverticulum is demonstrated and pushed into the wound by the esophagoscopist, who passes the esophagoscope through the mouth into the sac, which gives off a red glow from the light of the contained instrument. The sac is then grasped with nonlacerating forceps, while the esophagoscope, through which the sac has also been emptied and cleansed, is now advanced over the swallowed string by which it is guided through the subdiverticular opening deeper into the esophagus. The pouch is carefully liberated and its junction with the esophagus accurately defined by an upper and lower guide suture of fine silk. At this point a sterile duodenal tube is passed through the esophagoscope into the stomach. The esophagoscope is then withdrawn, leaving the feeding tube in place. The guiding thread is cut short to be later spontaneously passed through the intestine. Using the guide sutures as tractors, and with the wound protected by a layer of gauze, the neck of the sac is pulled into the wound and gradually divided with a follow-up continuous suture of fine black silk in a very fine curved eye needle. This suture to be both air- and water-tight should

be applied with about the fineness of an arterial suture. Each section of mucous membrane as exposed is carefully cleansed with small cotton swabs wet with 3.5 per cent tincture of iodine. The first row of sutures is inverted by two rows of interrupted no. 000 chromic catgut, or no. 36 alloy steel wire sutures placed in the outer wall of the hypopharynx and the overlying muscle. A 4-mm. split rubber tube drain is carried to the side of the esophagus below the suture line, to be removed at the end of forty-eight hours. The platysma is closed with interrupted 36 wire sutures tied in a square knot with ends cut very short, the skin with Michel's clips.

After Care.—The patient is placed in bed on a back rest. The feeding tube is brought out through the nose, by fastening its end to that of a lubricated 14 F. urethral catheter introduced to the pharynx through a nostril, and feeding started four hours after the operation provided the patient is awake and free from nausea. For the first twelve hours 60 cc. of a 10 per cent glucose solution is given every two hours. Nausea being absent the liquid is increased to 120 cc. every four hours. After forty-eight hours, from 180 to 240 cc. of liquids, including eggnog, modified milk, broths, fruit juices, thin gruels, and purées are given every three to four hours as borne. At the end of seventy-two hours the clips and drainage tube are removed. On the day following the patient may be permitted to be out of bed in a reclining chair. If there is no wound reaction, at the end of six days swallowing is tested by giving single teaspoonfuls of distilled water. If this does not cause distress or reaction, the feeding tube is removed the following day but only very thin liquids should be given for the next four or five days and solid foods only very gradually resumed after six weeks have elapsed. Should there be evidence of leakage into the tissues of the neck, as indicated by local tenderness, swelling, or redness with fever (in our experience a rare, almost unknown complication) the wound should be widely opened and drained and the duodenal tube feeding continued until the opening has closed.

W. WAYNE BABCOCK

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PART V. TRACHEA AND BRONCHI

APPLIED ANATOMY OF THE TRACHEOBRONCHIAL TREE FROM THE BRONCHOSCOPIST'S VIEWPOINT

The *trachea* deviates slightly to the right just below its entrance into the thorax. At the level of the second costal cartilage, the third in children, it bifurcates into the right and left main

aorta. This latter flattening is rhythmically increased with each pulsation. Under pathologic conditions, the tracheal outline may be variously altered, even to obliteration of the lumen. The mucosa of the trachea and bronchi is moist and glistening, whitish in circular ridges corresponding to the cartilaginous rings, and reddish in the intervening grooves,

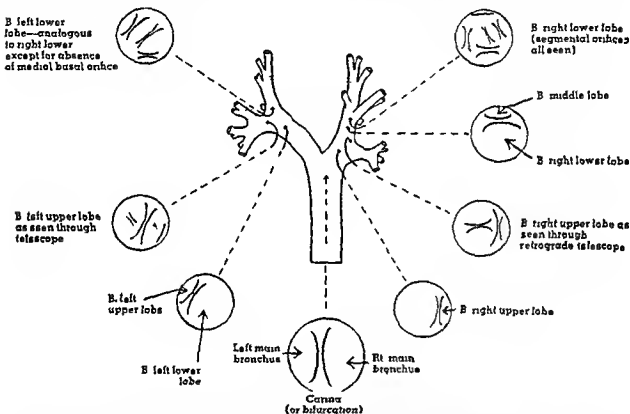


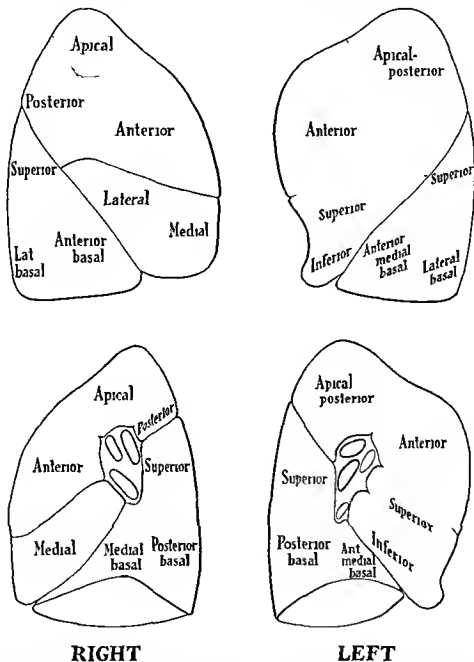
Fig. 437.—The lobar bronchi and their segmental branches, with schematic representation of the endoscopic landmarks. In this diagram the bronchial tree is shown 'upside down' because it is desired to represent the structures in the same relation as that which they bear to the bronchoscopist when the patient is examined in the usual position of dorsal recumbency.

bronchi. Posteriorly the bifurcation corresponds to about the fourth or fifth thoracic vertebra, the trachea being elastic, and displaced by various movements. The endoscopic appearance of the trachea is that of a tube flattened on its posterior wall. In two locations it normally often assumes a more or less oval outline—in the cervical region, owing to pressure of the thyroid gland, and in the intrathoracic portion just above the bifurcation, where it is crossed by the

The *right main bronchus* is shorter, wider, and more nearly vertical than its fellow of the opposite side, and is practically the continuation of the trachea, while the *left bronchus* might be considered as a branch. The deviation of the right main bronchus is about 25 degrees, and its length unbranched in the adult is very short, the proximal margin of its orifice is on a level with the carina. The deviation of the left main bronchus is about 75 degrees, and its adult

length is about 5 cm. The right main bronchus, considered as a stem, may be said to give off three branches: the *upper lobe bronchus*, the *middle lobe bronchus*, and the continuation

segmental bronchi, and the corresponding portions of the lobes which they supply may be called *bronchopulmonary segments* (Fig. 438). The right upper lobe is divided into three prin-



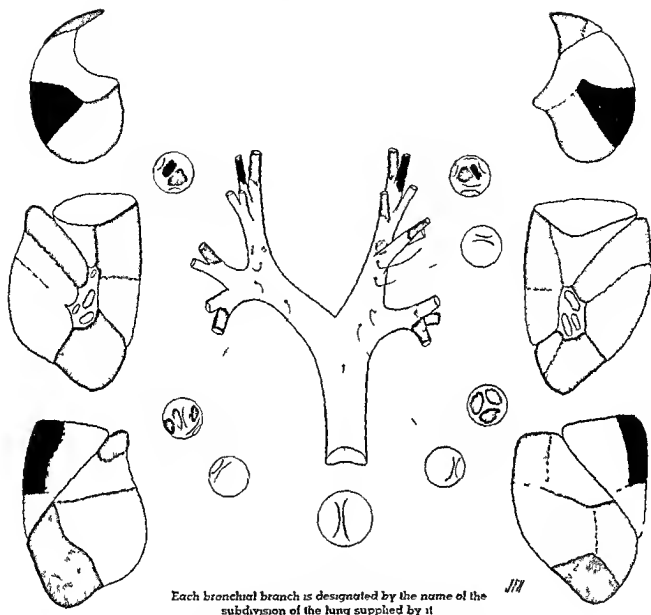
RIGHT

LEFT

Fig. 438.—The bronchopulmonary segments shown in both anterolateral (above) and medial (below) aspects. The terminology used in this illustration as well as in Figure 439 (*q v*) other illustrations and text is that suggested by Jackson and Huber.¹

downward called the *lower lobe bronchus* (Fig. 437). The left main bronchus gives off first the *left upper lobe bronchus*, and then continues downward as the *left lower lobe bronchus*. The branches of these lobar bronchi may be called

apical or *major segments* which are called, according to the terminology suggested by Jackson and Huber,¹ *apical*, *anterior*, and *posterior*, the middle lobe into two *medial* and *lateral*, and the lower into five *superior*, *medial basal*,



LEFT LUNG

LOBES

SEGMENTS

Upper	Upper Division	{	Apical posterior	<input checked="" type="checkbox"/>
			Anterior	<input type="checkbox"/>
	Lower (Lingular) Division	{	Superior	<input type="checkbox"/>
			Inferior	<input checked="" type="checkbox"/>
Lower		{	Superior	<input type="checkbox"/>
			Anterior medial	<input type="checkbox"/>
			Basal	<input type="checkbox"/>
			Lateral Basal	<input checked="" type="checkbox"/>
			Posterior Basal	<input checked="" type="checkbox"/>

RIGHT LUNG

LOBES

SEGMENTS

Upper	Upper	{	Apical	<input checked="" type="checkbox"/>
			Posterior	<input checked="" type="checkbox"/>
			Anterior	<input type="checkbox"/>
Middle	Middle	{	Lateral	<input type="checkbox"/>
			Medial	<input checked="" type="checkbox"/>
Lower		{	Superior	<input type="checkbox"/>
			Medial Basal	<input type="checkbox"/>
			Anterior Basal	<input type="checkbox"/>
			Lateral Basal	<input checked="" type="checkbox"/>
			Posterior Basal	<input checked="" type="checkbox"/>

Fig. 439 — Nomenclature for the bronchi and lungs suggested by Jackson and Huber¹ based on earlier terminologies of Kramer and Glass² Neil et al.³ and others

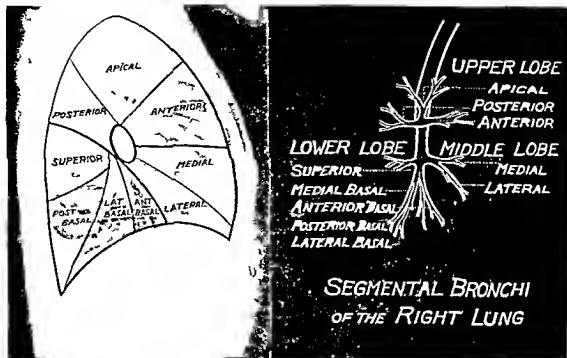


Fig 440—Segmental bronchi of right lung showing relation to bronchopulmonary segments in right lateral bronchogram (cf Fig 441)

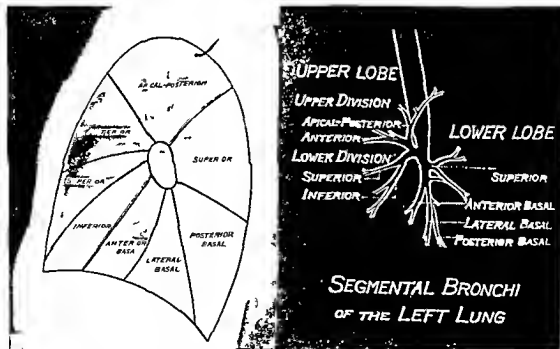


Fig 441—Segmental bronchi of left lung in relation to bronchopulmonary segments as shown in left lateral bronchogram (cf Fig 440)

anterior basal, lateral basal, and posterior basal. On the left side, the upper lobe is divided into an upper division and a lower lingular division

this latter portion of the left upper lobe being analogous to the middle lobe on the opposite side. The upper division of the left upper lobe

is divided into two major segments, the *apical* (or *apical posterior*) and the *anterior*, the lower



Fig. 442—Pulmonary abscess of anterior segment of right upper lobe as seen in right lateral projection

or "lingular" division is also divided into two segments, located one above the other, which

ments of the left lower lobe are, therefore *superior*, *anterior basal* (or *anterior medial basal*), *lateral basal*, and *posterior basal* (Fig. 439)

The septum between the right and left main bronchi, termed the "carina," is situated a little to the left of the midtracheal line. It is recognized endoscopically as a short, shining ridge running sagittally, or as the patient lies in the recumbent position we speak of it as being "vertical." On either side are seen the openings of the right and left main bronchi. In Figure 437, it will be seen that the carina is on a level with the upper portion of the orifice of the right superior lobe bronchus, with the carina as a landmark and by displacing with the bronchoscope the lateral wall of the right main bronchus, a second, smaller vertical spur appears, and a view of the orifice of the right upper lobe bronchus is obtained, though a lumen image cannot be presented. On passing down the right stem bronchus a horizontal partition or spur is found on the anterior wall, and anterior to this spur the lumen of the middle-lobe bronchus extends toward the ventral surface of the body. All below this opening of the middle lobe bronchus constitutes the right-

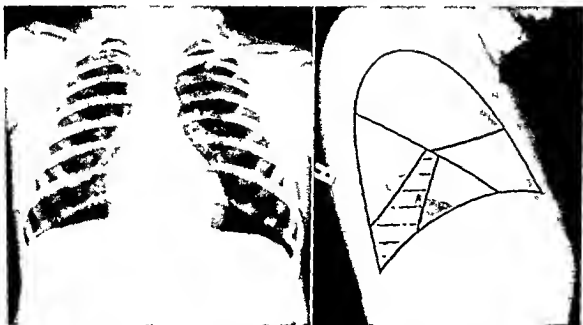


Fig. 443—Nail in the posterior basal segment of the right lower lobe bronchus

may be called the *superior* and *inferior* segments. The left lower lobe is divided exactly as the right, except that the medial basal segment forms a part of the anterior basal. The seg-

lower lobe bronchus and its branches. The first segmental orifice visualized in the lower lobe bronchus is that of the *superior segmental bronchus*, on the posterior wall, the next that of the

medial basal segmental bronchus, on the medial wall, and below these the orifices of the anterior basal, lateral basal, and posterior basal segmental bronchi.

Coming back to the carina and passing down into the left bronchus, the relatively greater distance from the carina to the upper-lobe bronchus than on the right side is noted. The spur dividing the orifices of the left upper lobe and lower-lobe bronchi is oblique in direction, and it is generally possible to see a little more of the lumen of the left upper-lobe bronchus than of its homologue on the right. Sometimes the spur dividing the upper from the lower (lingular) division can be seen. Below this are seen the lower lobe bronchus and its subdivisions (Fig. 437). The orifices of the segmental bronchi of the left lower lobe are analogous to those of the right, except for the absence of the orifice of a medial basal branch, this being a subdivision of the anterior basal segmental bronchus. While, as already mentioned, the orifices of the segmental branches of the upper-lobe bronchi are not ordinarily seen in routine bronchoscopy, they may be visualized with the aid of a *retrograde telescope* passed through the bronchoscope. The spur dividing the middle-lobe bronchus into medial and lateral segmental branches is likewise not ordinarily seen, but it can be seen in most adults by the introduction of a very small bronchoscope into the middle-lobe orifice.

In connection with the applied anatomy of the bronchi and lungs it should be noted that the lateral roentgen ray film is of special aid in not only the lobar but the segmental localization of a lesion or foreign body (Figs 440-443). It is hence obviously important for the bronchoscopist to have, in addition to anteroposterior (even though stereoscopic films) a good lateral projection as a preliminary study before undertaking endoscopic examination or treatment.

CHEVALIER L. JACKSON

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TECHNIC OF BRONCHOGRAPHY

The catheter technic described by Jackson and Bonnier¹ is, in the author's opinion, the most satisfactory of all methods of bronchography that have been devised. For routine bronchography in adults the ordinary soft catheter (16 Fr.) is best, and it is introduced through the mouth with a plain wire stylet bent to the "laryngeal curve" (Fig. 444), under the guidance of the laryngeal mirror. The preparation consists simply in keeping the patient fasting for a few hours before the procedure, and anesthetizing the pharynx and larynx by means of a spray of cocaine (4 per cent), or pontocaine (2 per cent), followed by a fractionated instillation of the same solution, using a laryngeal syringe under the guidance of the laryngeal mirror. In patients suspected of being sensitive to cocaine and pontocaine, larocaine (10 per cent) is substituted. After introduction of the catheter under guidance of the mirror, and withdrawal of the stylet, a small additional quantity of anesthetic solution is injected through the catheter, and then the patient is taken to the roentgen-ray department, the proximal end of the catheter being temporarily fastened to the lip and cheek with adhesive tape. The position of the tip of the catheter is determined behind the fluoroscopic screen, and if not satisfactory, it is corrected by appropriate manipulation of the catheter and the patient's head. Next, the oil is injected, either under fluoroscopic control or not, as desired, and the patient placed in various positions, according to the area that is to be mapped. In some cases the injection is made with the patient in lateral decubitus or in the "Cleopatra" position. If one base or any single lobe is to be mapped, 10 cc. of oil will suffice, but if both bases or a whole lung is to be mapped, not less than 15 to 20 cc. should be used. The author is of the opinion that it is best to remove the catheter quickly as soon as the injection has been completed, as in the great majority of cases definite removal of the catheter does not provoke the cough reflex, and the patient is much more at ease for any subsequent posturing, and for the taking of the pictures.

For bronchography in children the same type of catheter is used as in adults (in a size 14 Fr. instead of 16 Fr.), but the catheter is introduced through the nose and passed into the larynx either under the guidance of the direct laryngoscope or under fluoroscopic control. A semi-

rigid, woven catheter may be substituted for the soft one if the latter tends to be coughed out. If the direct laryngoscopic method is used it will be found surprisingly easy to direct the tip

tip of the catheter anteriorly into the larynx. It is remarkable how soon even an apprehensive child will become reconciled to the presence of the nasal catheter attached to his lip and cheek.

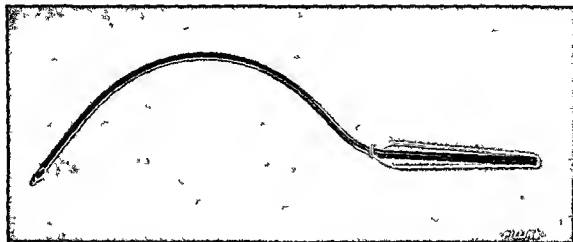


Fig. 444—Stylet-tipped catheter preferred by the author for the instillation of lipiodol for bronchography.

of the nasal catheter into the larynx, even without the use of forceps, by merely manipulating its proximal end. On the other hand it is also quite satisfactory, and less uncomfortable for

with adhesive tape. In order to help allay coughing a small amount of Iarocaine (10 per cent) may be instilled through the catheter, but cocaine is never used in children.

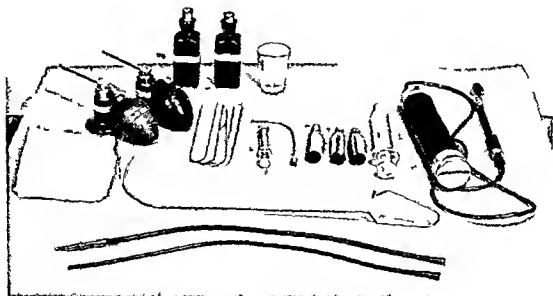


Fig. 445—Set up for lipiodol bronchography. Two atomizers and two bottles containing 10 per cent and 4 per cent cocaine respectively, medicine glass, tongue depressors, laryngeal syringe with curved tip, three ampules of lipiodol, 20-cc. syringe, stylet and two catheters, self-illuminated laryngeal mirror, 4 x 4-inch gauze.

the patient, to insinuate the catheter into the larynx under fluoroscopic guidance, tilting the head back just far enough to cause the curve of the posterior nasopharyngeal wall to direct the

Some have raised the objection to this technique that the passage of a catheter through the nose and into the tracheobronchial tree may carry infection from the upper to the lower air-

passages The author regards this objection as unsound, since it is well known that secretions from the nasopharynx pass freely into the

nic of Forestier and Leroux, in which the oil is washed back through the nose after successive washings with a special anesthetizing solution,

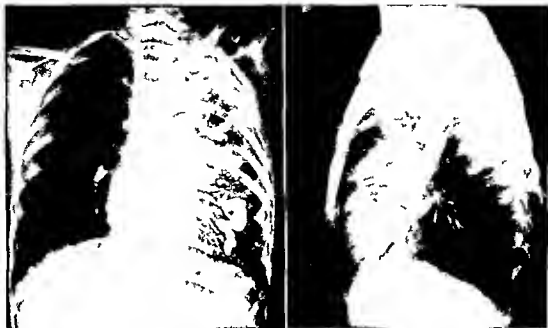


Fig 446—Multiple bronchiectatic abscesses of the upper lobe of the right lung Lateral projection localizes these cavities to the upper lobe and demonstrates normality of the lower lobe Lobectomy was subsequently done in this case

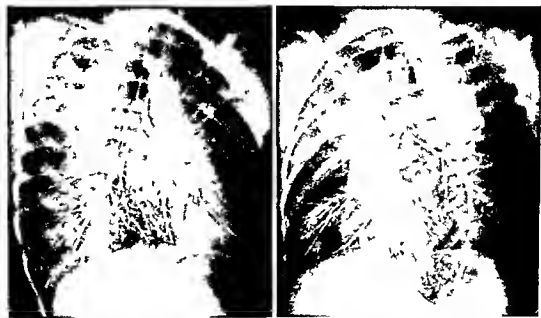


Fig 447—Right and left obliques should be taken if bilateral mapping is done at one sitting This was a case of chronic bronchitis without localizing signs Bronchography was done to rule out bronchiectasis

larynx and trachea during sleep, carrying down much more infection every night than could possibly be carried by the catheter This objection may apply to the use of the pernasal tech

especially in patients with a nose filled with purulent secretions, but we believe it is of no importance in connection with the catheter technic

When there is some reason for expediting the bronchography, in either adults or children, we introduce the catheter immediately on withdrawal of the bronchoscope after diagnostic bronchoscopy, with the aid of the direct laryngoscope. In this way the catheter method can be made to approximate one of the features very properly regarded by Clerf as an especial advantage of the bronchoscopic technic, namely, a combination of diagnostic bronchoscopy and bronchography in one procedure. The author is very strongly of the opinion, however, that as a rule much better and more uniform results are obtained if the oil is instilled through a catheter as has been described, rather than through the bronchoscope.

It is always preferable to map one lung at a time, because this permits us to obtain a good lateral as well as stereoscopic anteroposterior or postero-anterior views. The lateral projection is of great value to the bronchoscopist in the lobar localization of a lesion (Fig 446, and see also Figs 440 and 441), but it is of little use if both sides have been filled, because of the superposition of the images of the two sides. Both lungs may be injected at one sitting, however, if it is necessary to complete the study in one operation. In this case, stereoscopic anteroposterior and postero anterior views are taken, with the addition of right and left obliques (Fig 447). Right anterior oblique views are especially good in showing the lower "lingular" division of the upper lobe.

Regardless of the technic used, the wholehearted cooperation of the roentgenologist is necessary if good bronchographic work is to be accomplished. Not only must the quality of the roentgenography be of the highest, but the roentgenologist's aid in the posturing of the patient and in the choice of views to be taken to bring out the particular features of each individual case, is indispensable. As a matter of fact, in some institutions excellent bronchography is done by the roentgenologists themselves, but too often the roentgenologist does not have time or is not willing to use the catheter technic, and hence gets less satisfactory and certain results.

CHEVALIER L. JACKSON

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PHYSIOLOGY OF THE TRACHEOBRONCHIAL TREE

Tracheobronchial physiology is too large a subject for full consideration here. In addition to its obvious duties as a vital line of communication between the alveoli and the atmosphere, the tracheobronchial tree is equally important in peroral pulmonary drainage and in the maintenance of the acid base balance and the carbon dioxide tension in the blood (see under "Asphyxia"). Peroral pulmonary drainage¹ is dealt with on other pages. The air in the lungs of man normally contains about 5 per cent of carbon dioxide and about 14 per cent of oxygen, yet man lives in an atmosphere that ordinarily contains but a minute fraction of 1 per cent of carbon dioxide and about 20 per cent of oxygen. The need for oxygen in the tissues and the amount of carbon dioxide produced vary enormously with muscular activity, altitude, and other factors, yet carbon dioxide tension is maintained close to a 5 per cent standard, in order that man may ventilate his lungs proportionately to his need for oxygen and his expenditure of energy. The control is exerted through the carbonic acid content of the blood. Carbonic acid is very weak as an acid, and therefore can exist free in the blood which is alkaline, but the ratio between the two, called acid-base equilibrium, must be maintained within limits. Any alteration in the carbon dioxide content of the blood acts promptly on the respiratory center in the medulla, but the excitability of this bulbar center to the stimulus of the carbon dioxide is chiefly dependent upon the oxygen content of the blood and its relation to the quantity of blood alkali in use. In other words, there are two factors—the stimulus and the excitability of the center to the stimulus. The intensely interesting biochemical processes involved are clearly stated by Henderson.² The point of daily clinical importance to the bronchoscopist is that all of these vital processes are dependent upon a clear passageway for ingress and egress of air in the tracheobronchial tree. The responsibility of clearing obstructions from this vital passageway rests upon the bronchoscopist. He does it directly, mechanically, and efficiently. For the practical work of the bronchoscopist the most important movements of the bronchi are respiratory, pulsatory, and tussive. The latter are, essentially, exaggerated

respiratory excursions. The respiratory movements are an enlargement of lumen combined with an elongation, on inspiration, and a reciprocal diminution and shortening on expiration. The pulsatory movements are transmitted from the heart, aorta, and arterial trunks. The pulsatory movements, chiefly lateral, combined with the elongation and shortening of the respiratory excursions result in a wave like movement.⁴

CHEVALIER JACKSON

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BRONCHOSCOPY

The branch of medicine that treats of the tracheobronchial tree and its diseases is known as *bronchology*.

Bronchoscopy is the endoscopic examination and treatment of the tracheobronchial tree and lungs by means of the bronchoscope used as a speculum. Devised forty-odd years ago for the purpose of removal of inspired foreign bodies from the tracheobronchial tree, bronchoscopy has developed a new department of medical science based upon (1) the direct inspection of pathologic changes in the living bronchi, (2) the removal of specimens of tumor tissue and uncontaminated secretions of laboratory study, (3) improvement of drainage by (a) dilatation of stricture, (b) removal of obstructive tissues, granulomas, benign tumors, (4) the accurate localization of disease by serial examination of segmental bronchial orifices, (5) the treatment by direct application of chemotherapeutic agents to accurately localized foci of disease, (6) the aid given to the thoracic surgeon, (7) the reduction of incidence of postoperative pulmonary complications by preop-

erative and postoperative laryngoscopic aspiration in cases of prolonged operation.

The foregoing refers to present status. The future may add many diseases to the list, including pneumonia of the bacterial as well as the virus and other atypical forms.

Parallel achievements of the esophagoscope have established *esophagology* as a branch of medical science. The close clinical association between the two spheres of knowledge and activity have merged them into one branch called *broncho esophagology*.

Instruments and Equipment for Bronchoscopy.—The instruments for direct laryngoscopy were described and illustrated on a previous page. Additional instruments are necessary for bronchoscopy and esophagoscopy. To avoid the confusion that might result from two lists the laryngeal instruments are again listed here. An adequate equipment includes instruments of a number of sizes for adults, adolescents, children, and infants. The following list gives the sizes needed for all ordinary purposes.

- 1 laryngoscope (admitting 7 mm bronchoscope duplicates would save time in a busy clinic)
- 1 laryngoscope (child)
- 1 laryngoscope (infant)
- 1 laryngoscope anterior commissure
- 1 bronchoscope, 7 mm. by 40 cm. (general use, adults duplicates would save time in a busy clinic)
- 1 bronchoscope, 3.5 mm. (newborn) Holinger
- 1 bronchoscope 4 mm. by 30 cm. (infant)
- 1 bronchoscope 5 mm. by 30 cm. (child)
- 1 costophrenic inner bronchoscope, 4 mm. by 45 cm
- 1 esophagoscopic inner bronchoscope 3 mm. by 45 cm
- 1 esophagoscope, 5 mm. by 35 cm
- 1 esophagoscope 6 mm. by 35 cm. (child, full lumen)
- 1 esophagoscope 7 mm. by 45 cm. (full lumen, esophagoscopic bougienage)
- 1 esophagoscope 9 mm. by 45 cm
- 1 esophagoscope, 9 mm. by 53 cm
- 1 esophagoscope 9 mm. by 30 cm
- 1 extra light carrier and lamp for each lighted instrument *
- 4 yards worsted-covered cleaning wire
- 1 battery, triple circuit bronchoscopy (see note)
- 6 cords for battery, rubber covered
- 1 forceps side curved standard, 50 cm
- 1 forceps, rotation right angle 50 cm
- 1 forceps ring rotation, delicate, 40 cm
- 1 forceps pin medium light weight, 50 cm (Tucker)
- 1 forceps forward grasping medium, 50 cm (cupped)
- 1 forceps fenestrated, peanut, 40 cm
- 1 forceps, laryngeal grasping 28 cm
- 1 forceps, laryngeal cup, medium, straight

* George P. Pilling and Son Company of Philadelphia, who make these instruments, supply an extra light carrier and two extra lamps with each instrument. The extra light carrier tested and on the sterile table affords a quick replacement.

- 1 forceps laryngeal cup medium angular
- 1 forceps laryngeal cup delicate angular vocal nodule
- 1 forceps laryngeal tissue specimen 28 cm
- 1 forceps ball for tissue specimen esophageal 60 cm
- 1 forceps forward grasping 60 cm round tip
- 1 forceps, ring rotation fluoroscopic 60 cm
- 10 esophageal bougies nos. 10 to 28 F even numbers
- 6 sponge carriers bronchoscopic universa
- 2 aspirating tubes open end warning stop for 30 and 40-cm tube
- 1 aspirating tube open end 35 cm to go through 4 mm tube
- 3 aspirating tubes velvet eye 20 50 and 60 cm
- 2 aspirating tubes bronchial spiral flexible end one each straight and curved large
- 2 aspirating tubes bronchial spiral flexible end one each straight and curved small
- 2 specimen collectors 2 cc Lukens
- 2 boxes each bronchoscope sponges sizes 4 5 7 and 9
- 3 bite blocks 1 each child adolescent and adult C.L.J.
- 1 triple bronchoscope pump (see note)
- 1 bronchoscopic operating table with sponge rubber pad
- 1 stool for the bronchoscope st
- 1 stool revolving for assistant holding head
- 1 footrest for assistant holding head
- 1 bronchoscope instrument moisture proof cab net electrically warmed
- 1 bronchoscopic sterilizer on stand steam operated
- 1 battery table for triple batteries (see note)
- 1 basket for cords attached to battery table (see note)
- 1 copper case with rack for sterile instruments
- 1 Holman apparatus for wash ng tubular instruments

Note The pump triple batteries battery table and cord basket all combined in one unit (C.L.J.) will be found convenient (Fig 435)

In the following list are instruments that are useful for certain special purposes and should be included if likely to be unobtainable promptly when needed

- 1 laryngoscope (to admit 8 mm by 40 cm bronchoscope)
- 1 bronchoscope 8 mm by 40 cm
- 1 bronchoscope costophrenic 2 mm by 30 cm
- 1 bronchoscope costophrenic 3 mm by 35 cm
- 1 bronchoscope costophrenic 4 mm by 35 cm
- 1 bronchoscope (for deep bronchi of adult) 5 mm by 45 cm
- 1 laryngeal rotation forceps 28 cm
- 1 laryngeal basket punch forceps 28 cm
- 1 specimen collector Clerf with 2 glass cups

Selection of Tube for the Particular Case — The best guide is to look for a similar case as to size and location of foreign body and age of patient in the tables. It should always be remembered that a short tube prevents following the foreign body downward. There is no differ-

ence in the shape of a long and a short tube respectively. Therefore, unless an inner tube is to be used the tube selected should be long enough to follow the foreign body to the smallest bronchus it can enter or, in case of the esophagus, into the stomach.

Special Bronchoscopes — For some particular purposes special bronchoscopes are necessary. To reach a location near the periphery of the lung an instrument must be small enough to enter bronchi of small lumen and long enough to reach the location mentioned. An inside diameter of 7 mm is about the limit of practical work. These bronchoscopes are called costo-

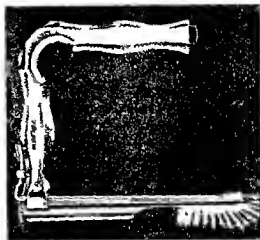


Fig 448 — Laryngoscope for introduction of bronchoscopes. The slide permit the removal of this necessarily rather heavy displacing instrument leaving the light and delicate instrument in the trachea for safe exploration of the tracheobronchial tree.

phrenic because they first came into use in the removal of pins and other foreign bodies of small diameter, like needles and pins that commonly invade the small branch bronchi in the costophrenic angle of the lung. They are passed through a larger bronchoscope and then are further used under guidance of the biplane fluoroscope. Other special bronchoscopes are mentioned in connection with the subject of foreign bodies in the air and food passages. A bronchoscope specially designed for electrocoagulation is useful in the treatment of some neoplastic and tuberculous lesions (Fig 485).

Laryngoscopes — For the introduction of bronchoscopes the laryngoscope with removable slide is used (Fig 448). It is necessarily of heavy strong construction to overcome the powerful muscles of the tongue, the pharynx, and some of those of the neck. Its removal

leaves the light and delicate bronchoscope (Fig 449) unencumbered for "gentle," harmless exploration of the tracheobronchial tree

electrically-operated negative pressure pump This pump is also incorporated in the unit just mentioned Specimen collectors of the Clérif or

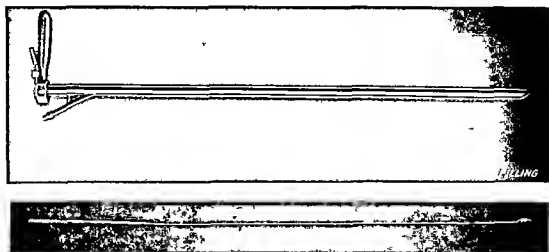


Fig 449—Bronchoscope made of thin metal, light and delicate for exploration of the tracheobronchial tree. It can be introduced with or without the laryngoscope. Its oblique lighting enhances definition. Below, A bronchial dilator for bronchoscopic use, made in ten sizes.

Aspirators—These are necessary for removal of tracheobronchial secretions for diagnosis and treatment. Our *aspirating tubes* (Fig 450) have an inlet especially designed to avoid trauma even in case mucosa should be drawn into them by negative pressure. The edges of the inlet are

Lukens type should always be sterile ready for immediate use regardless of the kind of case on the schedule.¹

Sponge Carriers and Sponges—The fundamental rule of surgery requiring constant maintenance of a dry field applies to bronchoscopy

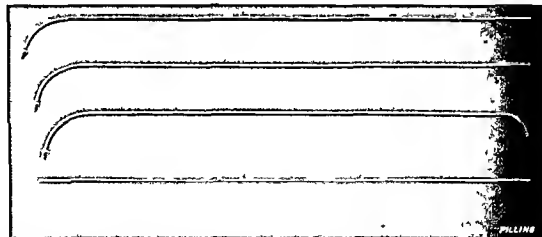


Fig 450—Aspirating tubes for use through the bronchoscope. Tubes with curved and flexible ends are used but are not necessary because the tussive squeeze forces secretions and exudates into the larger bronchial stems reachable with straight tubes. The lowermost instrument is a sponge carrier for carrying gauze sponges.

rounded inside and out and are depressed. Silk woven aspirating tubes are used for laryngoscopic aspiration, but not ordinarily for bronchoscopy (see under "Synergistic Bronchoscopic Aspiration of Pulmonary Abscess"). In use the aspirating tubes are connected with an

though it concerns secretions oftener than blood. The aspirator takes good care of maintenance of a dry field, yet sponging is occasionally necessary. For this purpose cotton-wrapped applicators should never be used. Sponges should be of folded gauze in four standard

sizes, a dozen or more of a size transfixed on a safety-pin, wrapped, and sterilized. These sponges are held securely in the sponge carrier illustrated in Figure 450. A few extra carriers avoid delay in removing and replacing soiled sponges.¹

Batteries—Commercial circuits should not be used for bronchoscopy because, no matter how perfect the theoretic protection, they involve a small degree of risk of electrocuting the patient who has the long moist contact of a bronchoscope, or esophagoscope, close to the nerves that control respiration and the heart. Storage batteries are sloppy, contain corrosives, and are otherwise inconvenient. Flashlight batteries are notoriously so unreliable that no conscientious endoscopist would think of depending upon them in a bronchoscopic operating room where the welfare and even the life of the patient calls for the utmost dependableness in equipment. Nothing equals the triple-circuit dry battery (Fig. 451) that for forty years has stood the test of dependable readiness twenty-four hours out of the twenty-four. Never once in all this time has the patient's welfare suffered for want of a light in the bronchoscope. To enable it to render this service, however, the battery was always given the simple but regular attention specified in the rules for its care.¹ It is incorporated in the combination operating room unit of Chevalier L. Jackson (Fig. 455).

Forceps—For bronchoscopy the best mechanism for forceps is the tube and stylet with hemostat type of handle. This construction gives slenderness of stem and delicacy of touch combined with great grasping power. Moreover, the jaws close in parallel position which favors retention rather than expulsion of the object grasped. Retention is also favored by backward canting of the serrations. The cant favors advancing of the jaws over a foreign body. For ordinary purposes forward grasping forceps are best, for specimens of tissue from the tracheobronchial tree the ball forceps serve well (Fig. 452). The forceps needed for various mechanical problems in foreign-body cases are described in connection with such problems. Before bronchoscopy the operator should make a final inspection and test of his forceps to see that the following adjustments are perfect: (1) that the jaws close properly, (2) that when the jaws are closed the upper halves of the handles just touch, (3) that the jaws open widely enough when open, and that they go into

the cannula as far as they should, when closed, (4) that they do not open too widely, especially in case of stiff expanding spring forceps, (5) that the end nut on the stylet is up to place, (6) that the side set-screw is up as tight as it will go with thumb and finger (not pliers), (7) that they work smoothly, if they do not, a little sterile petroleum jelly will help.^{1 2}

Bite Block—Wide gagging jams the mandible and hyoid bone, with their attachments, down over the larynx, and renders instrumental access and exposure of the laryngeal orifice difficult. All that is needed is some convenient means of preventing the patient from biting the tube. For this purpose nothing equals the thumb type of bite block to be used on the tip of

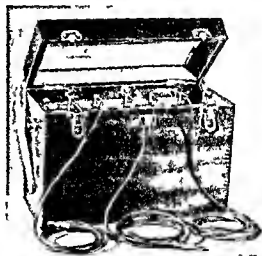


Fig. 451—Triple circuit battery for lighting laryngoscopes and bronchoscopes. A safe and dependable source of electric current. Dry cells, obtainable everywhere, are replaced every three months.

the index finger of the assistant who holds the head. Plastic or hard rubber are less uncomfortable for the patient when in contact with his teeth, as compared to the softest of metals (Fig. 455). In case of infants before eruption of any teeth the index finger alone is sufficient.

Dependableness in Equipment—Humanity and conscience require the utmost in dependableness in all equipment of an operating room dealing with the emergencies, such as impending asphyxia, that may be encountered in a bronchoscopic operating room. There are two fundamental rules for maintenance of this degree of dependableness: (1) All equipment must be in duplicate, as is obligatory in every commercial electric light plant. (2) All equipment should be closely inspected and tested after use,

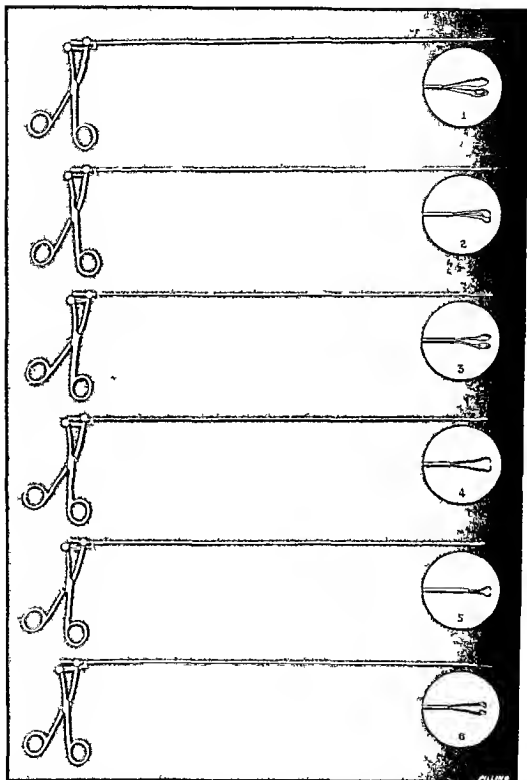


Fig. 452.—Forceps for bronchoscopy 1 Forward grasping forceps with serrated and slightly cupped jaws used for all ordinary purposes 2 Side-curved forceps also used for general purposes—the jaws are thin and flat 3 Ball forceps for taking specimens of tissue 4 Rotation forceps for holding securely while affording pivotal contact for changing a malpresentation to a favorable presentation 5 Ring rotation model that holds securely while permitting certain kinds of foreign bodies to dangle 6 Head holding forceps (C.L.J. model) for grasping head or shank of screws nails and tacks Special forms of forceps are required for special purposes

and if found imperfect it should be replaced by a duplicate while undergoing immediate repairs. This last rule means that the common custom of dumping all instruments in the sink for the inexperienced pupil nurse to clean and put away will not do for bronchoscopic equipment. Any instrument that is found bent must not be straightened thus risking breakage when used again possibly in an emergency. Any instrument found imperfect must be replaced by its duplicate while it is sent to the instrument maker for urgent and immediate repair. The rule of constant maintenance of duplicates must be always in the mind of the instrument nurse. The bronchoscopic operating room should be at all times set up sterile and covered ready for emergencies.

Tracheotomy Instruments—Tracheotomy is not necessary for introduction of a bronchoscope in any patient whose mouth can be opened yet patients in urgent need of tracheotomy arrive so often unexpectedly at a bronchoscopic clinic that the instruments for tracheotomy should be at all times kept sterile and properly wrapped ready for instant use (see under Tracheotomy.)

Oxygen Tank—The bronchoscopic room should always be equipped with an oxygen tank containing oxygen with carbon dioxide 7 per cent admixture. The just mentioned unexpected arrival of patients with impending asphyxia requires that a life saving equipment for bronchoscopic oxygen insufflation must at all times be sterilized and covered in readiness for instant

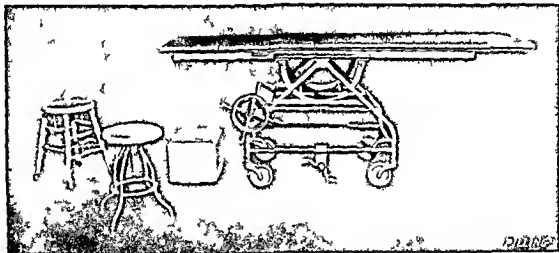


Fig 453.—Bronchoscopy table. This design has for many years served perfectly all the requirements of bronchoscopy, esophagoscopy and gastroscopy. The commonly used surgical tables are unsuitable for peroral endoscopy. Stools for endoscopy and head holder and also footrest for head holder are shown.

Lighting of Instruments—Overillumination of lamps should be avoided. It not only burns out the lamp but even more important it blanches the color of the mucosa in such a misleading way as to make inflammatory mucosa look normal. For standards of comparison the degree of illumination should be always the same. The proper point is reached when in turning up the rheostat the dull red of the filament begins to turn white. The dazzling brilliancy of the flash light should never be reached. The rheostat should always be started at zero lamps vary in resistance hence a proper setting of the rheostat for one lamp may overilluminate or burn out another lamp. The distally lighted bronchoscope (Fig 449) affords best delineation because of its oblique illumination.

use. It is most convenient as part of the unit (Fig 455).

Endoscopic Table—If a footrest of relative size as herein specified be provided any operating table may be used but the work is facilitated if a special table can be had which allows the placing of the patient in all required positions. The table illustrated in Figure 453 is so arranged that when the false top is drawn forward on the railroad the head piece drops and the patient is placed in the correct position for esophagoscopy or bronchoscopy i.e. with the head and shoulders extending over the end of the table (Fig 343). By means of the wheel the plane of the table may be altered to any desired angle of inclination or height of head. It cannot be too strongly stated however that the tilting

mechanism under control of the hand wheel is for use only *after* introduction of the bronchoscope, for introduction the table must be level except for special cases

Footrest—This is a very important part of the equipment. Makeshifts make trouble. We have used for many years a box (Fig. 453) specially made for the purpose. Its size and shape, 10 by 12 by 14 inches (25 by 30 by 35 cm.), give great stability and the choice of three heights, so that one can be selected that will be 56 cm. from the top of the table. This distance from the table top will be right for an assistant of average build (Fig. 343).

Spectacles—No one element in bronchoscopy is more important than utmost clarity of vision. If the operator has no refractive error he will need two pairs of optically plane, protective, hook-temple spectacles with very large "eyes." If he is ametropic, corrective lenses are necessary, if presbyopic the correction for direct laryngoscopy and bronchoscopy should be for 40 cm. focus. For esophagogastrosopy 60 cm. focus would be needed. Bifocal lenses are unsuitable for endoscopy, unless, of course, the operator trains himself to a special technic of looking. If lenses of two different foci are used the respective frames should be so different as to be instantly differentiated in subdued light. Whatever may be the glasses, plane or refractive, they must be in duplicate to avoid wasting time while the tube is in the patient. In many cases, pus, often bloody from granulations, is shot out of the bronchoscope. It would incapacitate the eye if glasses were not worn. The glasses, catching the secretion, need frequent cleaning and must be warm to prevent fogging when replaced. The clean spectacles should be put on instantly when needed, the soiled duplicates are cleaned with warm sterile water and kept warm in an ordinary electric heating pad ready for instant replacement on the operator. This care of protective glasses must be in charge of a nurse who has nothing else to do and she should have preliminary training in prompt taking off and putting on hook temple spectacles.

Conservation of Time—Time wasting during bronchoscopy should be eliminated. This does not mean that anything should be done precipitately, but poor organization is to blame when a baby with impending asphyxia is kept waiting while some forgotten item of equipment is searched for, or while an inexperienced

nurse takes the operator's soiled glasses out, washes, dries, and puts them on again, perhaps cold enough to be promptly fogged. After a bronchoscope goes down not a moment should be wasted, every moment is needed by the eye of the operator at the proximal end of the tube.

Light in the Bronchoscopic Room—The operator should work with both eyes open and with his right eye at the tube mouth. The operating room should be somewhat darkened so as to facilitate the ignoring of the image in the left eye, any lighting should be at the operator's back, and should be so subdued and so placed as not to cause reflections from the inner surface of his glasses. If the operator is left handed he may work with his left eye though he can educate his right hand so as to use his right eye, but right hand for forceps and left eye for vision or *vice versa* do not work well.

Color in the Bronchoscopic Room—The bronchoscopic operating room should be dull green in tone and to obtain this tone the walls, furniture, gowns, covers, towels, and all fabrics should be of approximately the same dull green color. The purposes of this are rest of the eye and increase of its power of perception of the endoscopic image. Most of these images are various nuances of red, therefore the green, combining red and blue, is complimentary as color and by contrast is not only restful to the eye but helpful in the appraisal of the tints of the various parts of the image. The customary glare of white was eliminated by substitution of green in our bronchoscopic room many years ago. The green has proved invaluable. Operators in all branches of surgery have found it advantageous for their work, and it is being generally adopted (Fig. 344).

Asepsis—Strict aseptic technic must be observed in all endoscopic procedures. The operator, first assistant, and instrument nurse must use the same precautions as to hand sterilization and sterile gowns as would be exercised in any surgical operation. The operator and first assistant should wear masks and sterile gloves. When placed on the table with neck bare and the shoulders unhampered by clothing, the patient is covered with a sterile sheet and the head is enfolded in a sterile towel. All of the tubes, and forceps are sterilized by boiling. The light carriers and lamps may be sterilized by immersion in 95 per cent alcohol or by prolonged exposure to formaldehyde gas. Continuous

sterilization by keeping them put away in a metal box with formalin pastilles, or other source of formaldehyde gas, could be practiced. Knives and scissors are immersed in 95 per cent alcohol, and the rubber covered conducting cords are wiped with the same solution, care being taken not to pull on them while doing so. It is to be remembered that while the patient is relatively immune to the bacteria he himself harbors, the implantation of different strains of perhaps the same type of organisms may prove virulent to him. Furthermore the transference of syphilis, tuberculosis, diphtheria, pneumonia, erysipelas, and other infective diseases

need for anesthesia even if the elimination of the voluntary muscles of respiration by the anesthetic were not mortal. Every member of the personnel must know his station, and everything he needs must be in precisely the same relation to his right and to his left hand respectively. The plan of stations and equipment worked out by the author in the early days of bronchoscopy and esophagoscopy has stood the test of time and the personal equation by hundreds of pupils. It is shown in the floor plan Figure 454. This set up must always be the same regardless of the character of the particular case.

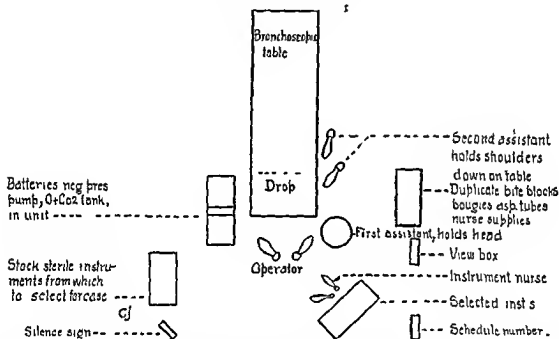


Fig 454.—Floor plan of the bronchoscopic operating room set up. To prevent confusion and time wasting and especially for prompt lifesaving efficiency the placing of all pieces of equipment and the stations of the personnel should always be precisely the same. Because of frequent emergencies such as the arrival of a patient with an impending asphyxia it is best to have the bronchoscopic room always set up with sterile instruments supplies and equipment. The unit indicated is shown in Figure 455.

from one patient to another would be inevitable if aseptic precautions were not taken. The simplest and only certain way is to carry out the standard operating-room sterile technic.

Introduction of the Bronchoscope—Orderly Procedure—To prevent confusion and dangerous delay in the desperate emergencies inevitably associated with the impending asphyxia and the almost moribund conditions of children arriving at a broncho esophagologic clinic, it is necessary to have an orderly routine established by daily following of systematized procedure. To attempt anesthetizing such patients would be almost inevitably fatal. There is no time nor

Anesthesia for Bronchoscopy—Unlike the larynx the bronchi are insensitive. Usually, the local anesthesia described for direct laryngoscopy (p. 436) is all that is required for any bronchoscopic procedure in adults. If desired, after the introduction of the bronchoscope a small quantity of a 4 per cent solution of cocaine may be applied to the bronchial mucosa of a particular region with a swab or atomizer, in adult patients. There is no pain from excision of a specimen of tissue. General anesthesia is not necessary for any bronchoscopic procedure. In children we use no anesthetic, general or local.

Technic of Introduction of the Bronchoscope — This procedure may be conveniently considered in two steps though in clinical work one step follows the other without a break

Step 2 Insinuation of the Bronchoscope through the Glottis The local anesthesia (in adults) for direct laryngoscopy is all that is needed for the introduction of the bronchoscope. The patient after the sermon on relaxation (qv) is placed in the position shown in Figures

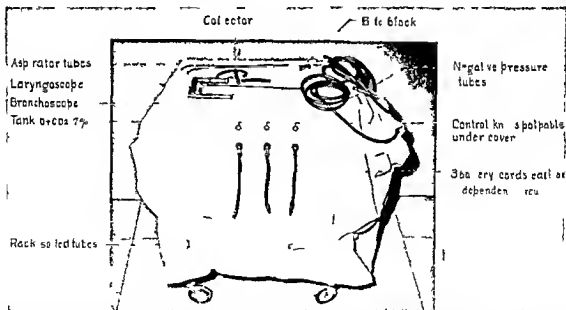
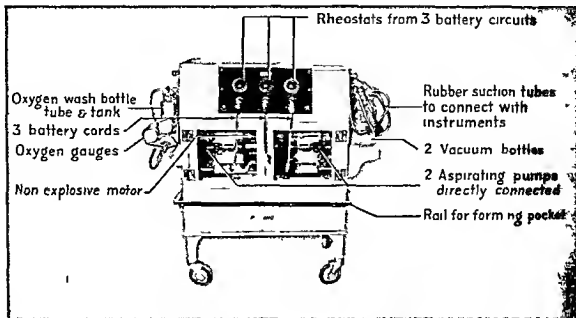


Fig 455 —Above Undraped unit Below Complete bronchoscope unit (CLJ design) set up and draped with sterile equipment ready for movement to any room in the hospital. In the bronchoscope operating room the instruments are on a separate table. The unit contains electric aspirating pump, triple battery for lighting endoscopic instruments, and oxygen-carbon dioxide tank. After each case the used tubing is dropped into the rack, not onto the floor, awaiting resterilization.

Step 1 Exposure of the Larynx to View Except that the laryngoscope is of the slide type every detail of this step is precisely the same as described under the head of "Direct Laryngoscopy" and illustrated in Figures 342, 343, and 344 (pp 433, 434, 435).

342 and 343 with head and shoulders projecting over the edge of the table and the head supported 15 cm above the table level by an assistant. The glottis is exposed by left-handed laryngoscopy, the steps of which are shown in Figure 457. The instrument nurse is

holding the bronchoscope lighted with its own cord with handle out to the right in position for insertion. The operator now grasps the bronchoscope, his eye is transferred from the laryngoscope to the bronchoscope and the bronchoscope is advanced and so directed that a good view of the glottis is obtained. The distal end of the bronchoscope should then be directed to the left so as clearly to expose the left cord. In this position it will be found that the tip of the slanted end is in the center of the glottic chink and will slip readily into the trachea. No great force should be used because if the bronchoscope does not go through readily the tube is either too large a size or it is not correctly placed. Slight rotation of the bronchoscope may help. Normally however there is some slight resistance which in cases of subglottic laryngitis may be considerable. The trained laryngologist

that the axis of the bronchoscope corresponds to the axis of the trachea in order to avoid injury to the subglottic tissue which might be followed by subglottic edema (q.v.). If the subglottic region is already edematous and causes resistance, slight rotation to the laryngoscope and bronchoscope will cause the bronchoscope to enter more easily.

Difficulties in the Introduction of the Bronchoscope.—The beginner may introduce the bronchoscope into the esophagus instead of the trachea; this might be a dangerous accident in a dyspneic patient not only by the delay but also because the tube could occlude the trachea by compression and thus cause asphyxia. If the



Fig 456.—Direct laryngoscopy, recumbent patient. The first assistant is sitting holding the head as shown in Figure 343. The second assistant is holding down the shoulders. The fingers of the operator's right hand pull the upper lip out of danger of getting pinched between the teeth and the laryngoscope. This is a precaution of the utmost importance and the trained habit of doing it must be developed by the peroral endoscopist. The instrument nurse is holding the bronchoscope in proper position for entering the tube with its handle out to the right.

gist will readily determine by sense of touch the degree of pressure necessary to overcome it. When the bronchoscope has been inserted to about the second or third tracheal ring, the heavy laryngoscope is removed by (a) rotating the handle to the left, (b) removing the slide and (c) withdrawing the instrument, which is (d) taken by the instrument nurse who (e) replaces the slide in readiness for immediate use if needed again (see Figs 457, 458, 459, 460). Care must be taken that the bronchoscope is not withdrawn or coughed out during the removal of the laryngoscope; this can be avoided by allowing the proximal end to rest against the gown-covered chest of the operator. At the moment of insertion of the bronchoscope through the glottis, an especially strong upward lift on the beak of the spatula will facilitate the passage. It is necessary to be certain

given technique be carefully carried out the bronchoscope will not be contaminated with oral secretions. The trachea is recognized as an open tube, with whitish rings; the expiratory blast can be felt and tubular breathing can be heard, whereas, if by mistake the bronchoscope has entered the gullet, the cervical esophagus will be recognized by its collapsed walls. A puff of air may be felt and a fluttering sound may be heard when the tube is in the esophagus, but these lack the intensity of the tracheal blast. Usually a free flow of secretion is met with in the esophagus. In diseased states the tracheal rings may

not be visible because of swollen mucosa, or the trachea itself may be in partial collapse from

greatest difficulties in the introduction of the bronchoscope are in the exposure of the larynx

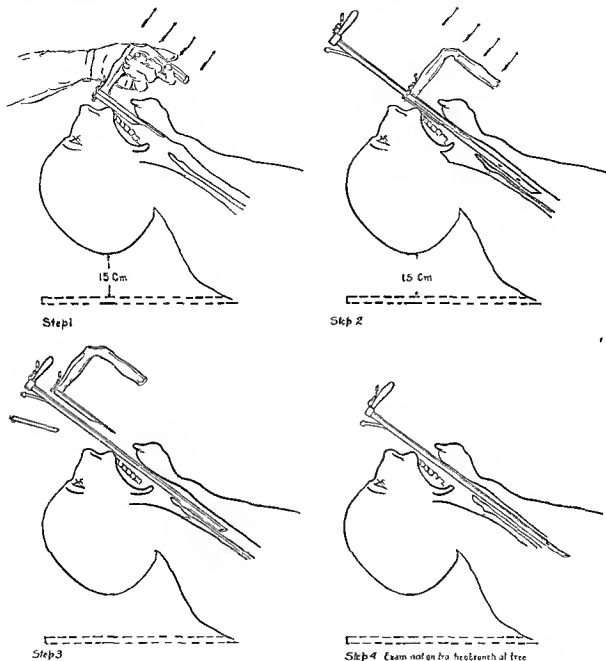


Fig. 457—Schema illustrating standard technique of bronchoscopy. The portion of the table here shown under the head is in actual work dropped all the way down perpendicularly. It appears in these drawings as a broken line to emphasize the fact that the head must be above the level of the table during introduction of the bronchoscope into the trachea. Step 1, The larynx is exposed by a lifting motion as indicated by the parallel darts. Step 2, The bronchoscope is introduced through the laryngoscope. Step 3, The heavy laryngoscope is removed by withdrawing the slide. In the lowermost illustration the light delicate bronchoscope is in the trachea free of all encumbrance for gentle manipulations and search without danger of injury to the delicate tracheobronchial tree even that of a baby.

external pressure. The true expiratory blast will, however, always be recognized when the tube is in the trachea, if the patient is breathing. The

These difficulties are fully described in connection with the subject of direct laryngoscopy, which should be carefully and frequently studied



Fig. 458.—Insertion of the bronchoscope. Note direction of the trachea as indicated by the bronchoscope. The operator, having exposed the glottis with the laryngoscope in the left hand, has now transferred his eye to the bronchoscope which he is insinuating in the glottic chunk. Note the handle of the bronchoscope is to the right and is held lightly, not grasped in the clenched fist (Fig. 462) At this time the instrument nurse has stepped aside toward the left ready to receive the laryngoscope from the operator's left hand.



Fig. 459.—The bronchoscope is now deep in the trachea (Step 2, Fig. 457). The instrument nurse, who has stepped aside for the taking of the photograph, will take from the operator the rubber tube and attach it to the independent aspirating specimen collector.

It must always be remembered that the bronchoscope is *insinuated* not forced, through the larynx

Examination of the Trachea and Bronchi—Every landmark and all bronchial orifices must be identified *seriatim* because this is the only way by which the bronchoscopist can know what part of the tree he is examining. From the moment the tube mouth passes the glottis the operator must know at every moment precisely where the tube mouth is. Appearances alone are not enough. It is the order in which they are

be similar to that of holding a pen—that is, the thumb, first and second fingers encircle the shaft of the tube. The bronchoscope should never be held by the handle (Fig. 462). Such a grasp does not allow of tactile sense transmission—it is rigid, awkward, and it renders rotation of the tube a strong wrist motion instead of a gentle finger action. Any secretion in the trachea is to be removed by aspiration before the bronchoscope is advanced. The inspection of the walls of the trachea is accomplished by weaving the bronchoscope from side to side.



Fig. 460—The heavy laryngoscope has been removed, leaving the light bronchoscope in position. The operator has inserted the aspirator. Note how the left index finger of the operator retracts the upper lip while the tube is held lightly between the thumb and second fingers of the left hand; the last two fingers are hooked over the upper teeth of the patient, anchoring the tube mouth to prevent it moving in or out or otherwise changing the relation of the distal tube mouth to a foreign body or a growth while the forceps are being used. Thus also any desired location of the tube can be maintained in systematic exploration. The handle has been turned to the right to enter the right bronchus.

exposed that enables the inexperienced operator to know the orifices. After the removal of the laryngoscope the bronchoscope is to be held by the left hand like a billiard cue; the terminal phalanges of the left middle and ring fingers hooking over the scope, clamping it to the teeth tightly or loosely as required (Figs. 460, 461). Thus the tube may be anchored in any position or at any depth, and the right hand which was directing the tube is free to be used for the manipulation of other instruments. The grasp of the bronchoscope in the right hand should

and, if necessary, up and down; the head being deflected as required during the search of the passages, so that the larynx is not made the fulcrum in a lever-like action.

In passing down the trachea the carina must be identified. Before entering either main bronchus the orifices of both should be identified and inspected. The *carina* is identified as a sharp, nearly vertical spur (recumbent patient) at the distal end of the trachea, on either side of it are the openings of the main bronchi. As the carina is situated to the left of the midline

of the trachea the lip of the bronchoscope should be turned toward the left and slight lateral pressure should be made on the left tracheal wall while the head of the patient is held slightly to the right. This will expose the left bronchial orifice and carina. If this be not done the bronchoscope will pass down the right bronchus and the operator may not see the left bronchial orifice or even the carina.

ENTERING THE BRONCHUS—The lip of the bronchoscope should be turned in the direction of the bronchus to be explored and the axis of the bronchoscope should be made to correspond as nearly as possible to the axis of this bronchus. The position of the lip is designated by the direction taken by the handle. Upon entering the right bronchus the handle of the bronchoscope is turned horizontally to the right and at the same time the assistant deflects the head to the left.

The *right upper lobe bronchus* is recognized by its vertical spur; the orifice is exposed by displacing the right lateral wall of the right main bronchus at the level of the carina. Usually this orifice will be thus brought into view. If not the bronchoscope may be advanced downward 1 or 2 cm. taking care to avoid overriding. This branch is sometimes found coming off the trachea itself and even if it does not the overriding of the orifice is certain if the right bronchus is entered before search is made for the upper lobe bronchial orifice. The head must be moved strongly to the left in order to view the orifice. A lumen image of the right upper lobe bronchus is not often obtainable because of the angle at which it is given off and the shortness of its stem. To get a good view of this orifice it is generally necessary to use the retrograde telescope passed through the bronchoscope.

The *left upper lobe bronchus* is entered by keeping the handle of the bronchoscope (and consequently the lip) to the left and by keeping the head of the patient strongly to the right as the bronchoscope goes down the left main bronchus. This causes the lip of the instrument to bear strongly on the left wall of the left main bronchus; consequently the left upper lobe bronchial orifice will not be overridden. The spur separating the upper lobe bronchial orifice from the stem bronchus is at an angle approximately from two to eight o'clock as usually seen in the recumbent patient. A lumen image of the lower or lingular division of the upper

lobe bronchus is often obtained if the patient's head be borne strongly enough to the right.

The orifices of the segmental branches of the lower lobe bronchus in either lung are exposed or their respective lumina presented by manipulation of the lip of the bronchoscope with movement of the head in the required direction. Posterior branches require the head quite high. A large one in the left stem just below the left upper lobe bronchus (the anterior basal branch) is often invaded by foreign bodies. Anterior branches require lowering the head. The *middle lobe bronchus* is the largest of all anterior

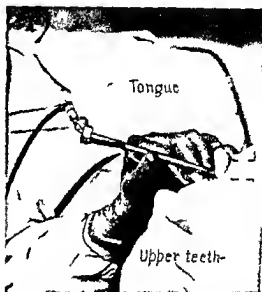


Fig. 461.—Position of the left hand for fixing the bronchoscope in a desired relation to a lesion or foreign body for study or use of forceps. Fixation must be only gentle. It must yield to strong tussive movements but if not fixed at all the elongating and shortening respiratory movements of the tree would shift the bronchoscope about with a resultant panoramic succession of views and temporary loss of the desired image. One soon learns to allow the tube to go up and down with the respiratory and tussive movements yet retain approximately the same relation to the object.

branches. Its almost horizontal spur is brought into view by directing the lip of the bronchoscope and dropping the head of the patient until the lip bears strongly on the anterior wall of the right bronchus. Otherwise the middle-lobe bronchial orifice will be passed without being seen. With this general over-all grasp of the endoscopic principles and with a few landmarks firmly fixed in mind the examiner will have pegs on which to hang the essential studies of the applied anatomy of the tracheobronchial tree and lungs as given in connection with that subject (See p. 597 et seq.).

The bronchoscopist must know the segments of lung to which each branch bronchial orifice leads. This knowledge can be acquired only by an anatomic study including (1) the chart showing the segments, (2) a corresponding model, (3) a cadaver whose chest is open to

bronchoscope or it may be passed through the latter after the orifice of the segmental or small bronchial branch has been reached. The endoscopist looks alongside the tube not through it as it is passed through the laryngoscope or bronchoscope. The light of the latter illuminates



Fig. 462—At the left is shown an incorrect manner of holding the bronchoscope. The grasp is too rigid and the position of the hand is awkward. At the right is shown the correct way of holding a bronchoscope (or esophagoscope) with the right hand. The thumb must not occlude the tube mouth. During most of the work with the bronchoscope and esophagoscope the collar of the proximal end is held about as one would the ocular end of a small telescope. The "handle" is of use as a protector of the light-carrier connection and, most important, as a subconsciously-used index of the lip of the tube mouth.



Fig. 463



Fig. 464

Fig. 463—Introduction of the bronchoscope without the laryngoscope. The position of the patient's head is the same as in the standard technic, high at first. The operator is in the same standing position. The correct holding and poise of the bronchoscope are here shown.

Fig. 464—Introduction of the bronchoscope without the laryngoscope. As the bronchoscope passes down the trachea the head is lowered.

manipulation by removal of the anterior wall, and (4) the relations of each bronchial orifice as seen through a bronchoscope inside the tracheobronchial tree of the cadaver.³

Introduction of the Costophrenic Bronchoscope.—The costophrenic bronchoscope may be introduced in the same way as the standard

the field during passage. Thereafter the guidance is altogether by the biplane fluoroscope. (See the section on pins at the periphery of the lung.)

Introduction of the Bronchoscope without the Laryngoscope.—In the foregoing pages the standard technic for bronchoscopy has been

given. It should be thoroughly mastered by everyone who wishes to do bronchoscopy. Mastery can be accomplished by, first, routine drill on the cadaver, then by bronchoscopies on hundreds of patients. Having thoroughly mastered it the bronchoscopist may qualify himself to introduce the bronchoscope without the laryngoscope.

In this optional technic (Fig. 463) the bronchoscope is grasped lightly but firmly in the right hand, like a pen, and steadied with the left hand as it is passed back over the tongue, a little toward the right angle of the mouth. As the tongue is lifted forward the edge of the crest of the epiglottis is visualized, the tip of the bronchoscope is then passed under the epiglottis and the epiglottis is lifted forward. The glottis is visualized as a black opening bordered on each side by the vocal cords and posteriorly, by the posterior commissure. If the larynx is well anesthetized the cords will remain in such complete abduction that they are not conspicuous. If local anesthesia is insufficient the glottis may close in spasm, in which case the scope is rotated slightly as the bevel tip is insinuated through the glottic chink. In any case great care must be taken not to damage the arytenoid cartilages or posterior commissure. If the anesthesia is satisfactory and the patient well relaxed the bevel tip should be directed forward during introduction, which is indicated, of course, by the corresponding anterior position of the handle (Fig. 464). Just as in the introduction through the laryngoscope, the head is elevated and slightly extended at the start, and as soon as the bronchoscope has passed into the trachea, the head is lowered.

There has been a considerable amount of discussion relative to the advantages and disadvantages of the above described technic. For example, it has been said that, by this technic, contamination of the trachea and bronchi with oral secretions is likely to be produced. This is quite possible, and especially so if difficulty is encountered and the tip of the bronchoscope is passed into the tonsillar fossa or up and down the posterior pharyngeal wall, scooping up a mass of secretion. In such cases it is best to resort, without delay, to the use of the laryngoscope to expose the larynx (using, of course, another bronchoscope). If the bronchoscope is introduced easily and directly into the glottis, however, there is little likelihood that there will be carried into the tracheobronchial tree more in the way of infective agents than can promptly be annihilated by the defensive power of the lungs against infective invasion through the epithelial barrier of the endobronchial mucosa. This barrier is, of course, most efficient against any infection carried in the patient's throat, and to which he therefore has developed a partial immunity. There should be no relaxation of the

sterile operating-room technic to guard against infections from preceding patients and especially against the specific infections such as syphilis, tuberculosis, diphtheria, and the exanthemas. Among the advantages of this simplified technic must be mentioned the lessened discomfort for the patient, the greater speed of introduction, and the simplified technic for the instrument nurse.

It should be emphasized, however, that no one should use this optional technic until he has become thoroughly familiar with the standard classical technic, using the laryngoscope. He should have become thoroughly familiar with the endoscopic appearance and relations of the epiglottis, aryepiglottic folds, ventricular bands, ventricles, true cords, and subglottic region, as well as the pyriform sinuses and esophageal orifice, as seen through the laryngoscope. Furthermore, we believe that it is best to restrict this technic to older children and adults, invariably using the laryngoscopic technic for infants and young children.

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BRONCHOSCOPY IN RELATION TO OPERATIVE SURGERY

It is a platitude to say that the life of any patient depends upon maintenance of that vital "line of communication," the airway to the pulmonary alveoli. Up until recent years about all the anesthetist had for clearing this airway was a tongue forceps, a gauze swab for the pharynx, and his two hands used externally for lifting the tongue forward at the angle of the mandible. The anesthesiologist of today has at his command a direct laryngoscope, broncho-

scope, aspirator, and various tubular means for maintaining a clear passage for air, and to the air he adds anesthetic vapors, if an inhaled anesthetic is desired. If other than an inhaled anesthetic is needed he attends to that and, in addition to his watch on the heart and circulation, he sees to it that the airway is always clear, before, during, and after operation, by synergistic aspiration with the silk-woven aspirating tube inserted through the laryngoscope (Figs 469, 470). Particularly he has charge of that most dangerous factor, premedication. The risks in the use of those annihilators of the cough reflex, morphine and atropine, are not yet fully realized, though it was shown over forty years ago that they are a large factor in the causation of postoperative pulmonary complications.^{1 2 3 4 5}

Bronchoscopy in Relation to Thoracic Surgery.

—The foregoing paragraph refers to general operative surgery. Modern thought is that the surgeon who specializes in thoracic surgery should also be a bronchoscopist.^{1 2 3 4} This, however, is not enough. He needs additionally an assistant who is a bronchoscopist and he should have an anesthetist who is a bronchoscopist, though the latter two may be combined in one individual, if desired. Thoracic surgeons find the bronchoscopic studies in normal and morbid anatomy interesting and helpful, but at the operating table the thoracic surgeon, apart from vital time loss, cannot leave his sterile field to clear the patient's tracheobronchial airway. This duty today devolves upon the bronchoscopically trained assistant or similarly trained anesthetist.² The subject of diagnosis is considered on other pages. *Preoperative aspiration* is important.^{1 2 3 4} Preparatory to operation in any case of suppurative pulmonary disease, clearing of the tracheobronchial tree of accumulated pus not only gives a start with a clear channel preventive of asphyxia or anoxia, but, equally important, it removes the material from which postoperative plugs could be formed. Such material may reaccumulate during operation, but it certainly is better not to start with an accumulation. In case of larger accumulations preoperative bronchoscopic aspiration prevents manipulative dumping of purulent collections from a suppurative focus in one lobe or lung into a sound lobe or lung upon whose respiratory gaseous exchanges the patient's life depends. Abscess is not the only condition to which this applies, bronchiectatic

accumulations are in some cases large enough to drown two sound lobes. Postural drainage is good and should be used but it is not sufficient to empty the ramifications and strictured passages of a bronchiectatic lobe. Preoperative aspiration may be done in bed, in the preparation room, or on the operating table, as the surgeon may deem best. It should be done without any anesthetic that would suppress the cough reflex for reasons herein stated in connection with "Synergistic Bronchoscopic Aspiration of Pulmonary Abscess," and "Laryngoscopic Aspiration." During operation the bronchoscopic assistant has the bronchoscope either in the patient or ready for immediate insertion if needed. Thus perfect control of the airway throughout the operation prevents asphyxia and even moderate anoxia. Prolonged anoxia is a powerful depressant and prolonged excessively high carbon dioxide tension fatigues the respiratory centers. In the surgical care of injuries, accidental or military, involving the thoracic cage or its contents, bronchial obstruction is one of the dangers to be promptly discovered and quickly relieved by bronchoscopy.^{1 2 4 5} The obstruction may be by inspired material, blood, reaction, inflammatory swelling or exudates, compression, or lesions of bronchial or parenchymal tissue. Areas of atelectasis may be the cause or the effect of bronchial obstruction. Injuries to the bronchi and parenchymal pulmonary tissue may be indicated by blood issuing from the mouth or by blood-streaked sputum, but these signs may be absent. Undoubtedly in all cases of trauma of the chest wall, however slight, and whether accidental, surgical, civil, or military, the patient should be watched for roentgen-ray and physical signs of bronchial obstruction, so that bronchoscopic aid to the surgeon in diagnosis and treatment may be promptly afforded.^{1 2 3 4 5} In cases of thoracotomy for disease *postoperative bronchoscopic aspiration* is important. Utmost bronchoscopic vigilance is necessary to make sure that the bronchial tree on the sound side is draining and ventilating the good lung on which the patient's life depends. These and other phases of after care are considered in connection with bronchial obstruction (see, particularly, paragraphs on "Cough," and "Synergistic Bronchoscopic Aspiration of Pulmonary Abscess"). In all of the foregoing statements only bronchoscopic aspiration is mentioned. It should be understood that, in patients

whose tracheobronchial tree has been thoroughly studied bronchoscopically preoperative and postoperative aspirations can be done simply by laryngoscopic aspirations with the silk woven aspirating tube (Figs 468 469 470) without the bronchoscope All anesthesiologists are now trained to do this work

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BRONCHOSCOPY IN DISEASES OF THE TRACHEA BRONCHI AND LUNGS

Broadly stated the bronchoscope bears the same relation to intrathoracic disease as the vaginal speculum does to pelvic disease Specular examination anywhere may reveal (1) a normal state (2) a condition amenable to treatment through the speculum (3) a condition amenable to treatment by irradiation or physical therapy or (4) a condition that should be dealt with by external surgery¹ Obviously therefore to endeavor to state the *indications for bronchoscopy in disease* would seem as absurd as to ask a gynecologist to state the indications for use of the vaginal speculum

Specular examination is an essential part of a complete examination of any part of the human body for which a speculum is available In dealing with intrathoracic disease the internist can tap and look and listen on the outside the roentgenologist can in a sense look through the patient the bronchoscopist can look inside the lungs and can bring up specimens of tissue and uncontaminated secretions for the laboratory Here we have a diagnostic team never before equaled for coping with pulmonary disease The surgeon brings to the group a ripe experience in the treatment of pulmonary suppuration The internist with his broad viewpoint will serve as a balance wheel Surgical cases will be treated by the surgeon medical cases by the internist and they will add peroral bronchoscopic drainage and bronchoscopic relief of bronchial obstruction to their other therapeutic measures in suitable cases For the treatment of inspired foreign body (*qv*) bronchoscopic methods have the unique position that renders no other method worthy of a moment's consideration² With the sole exception of this class of case the bronchoscopist is merely an assistant to the physician and surgeon It may however be stated in a general way that there is one class of case in which full justice is not done the patient if diagnostic bronchoscopy be omitted and that is any case showing physical or roentgen ray signs of *bronchial obstruction* (*qv*) It may be added that the occasional occurrence of obstruction without any physical or roentgen ray signs of such a lesion renders it more accurate to state that bronchoscopic examination is indicated in every patient with any pulmonary symptom¹

Contraindications to bronchoscopy in disease do not exist if the bronchoscopy is really needed Serious organic disease such as aneurysm hypertension or advanced cardiac disease might render bronchoscopy inadvisable except for the removal of foreign bodies Acute respiratory infections such as the common cold may render postponement of bronchoscopy advisable yet bronchoscopy is urgently indicated in acute laryngotracheobronchitis (*qv*) There are many other paradoxical circumstances that prevent definite statements of indications and contraindications For example a moderate hemorrhage from the lungs might be a good reason for postponing a bronchoscopy yet in case of a severe hemorrhage bronchoscopic hemostasis might be needed Hemoptysis (*qv*)

is almost always an indication for bronchoscopy to determine the source and character of the lesion concerned

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BRONCHIAL OBSTRUCTION

Any morbid diminution of the lumen of a bronchus is known as a bronchial obstruction. Probably no human being attains maturity without having had bronchial obstruction in some form and in some degree.

Etiology and Pathology.—The contributory causes are so numerous that only the most important can be mentioned. *Infection*, primary or secondary, specific or nonspecific, participates in causation in most cases. *Inflammation*, congestion, or any morbid process that thickens the lining mucosa and submucosa diminishes the lumen, and this diminution is in geometric progressive proportion inversely as the diameter of the lumen of the affected bronchus. For rough illustration, it may be said that in a bronchus of 10-mm interior diameter a swelling increasing the thickness of the mucosal lining 1 mm will decrease the lumen to 8 mm, which is 20 per cent. In a bronchus of 2-mm diameter, thickening that approaches 1 mm at the periphery of the lumen will obliterate it, which is to say 100 per cent.¹ This brings *oedema* into consideration. It is natural to think that a small baby needs only a small bronchus, which is true only when there is no obstruction. In any obstructive bronchial condition the baby is at a frequently fatal disadvantage in three ways namely, (1) small diameter of bronchial lumen, (2) weak *bechic* blast,¹ and (3) weak *tussive* squeeze.² These are the usually ignored physical factors that explain the fact that pulmonary complications are the terminal phase of most deaths in children. Weak *tussive* squeeze and *bechic* blast along with edematous obstruction from hypostatic engorgement may be considered the usual contributory factors in old age

Secretions, *exudates*, and all morbid products dumped into the bronchi for disposal by the mechanism of *peroral pulmonary drainage*² are factors of first importance in bronchial obstruction. This natural drainage system consists of (1) *the cilia*, (2) *the tussive squeeze*, (3) *the bechic blast activated by* (4) *the cough reflex* are.² Considering the fundamental importance of this drainage and defensive mechanism it is appalling to contemplate the fact that one of the greatest medical mistakes in all history was the therapeutic antagonism to cough.³ For centuries cough was regarded as a pernicious, distressing reflex to be suppressed by *opium* and in the nineteenth century *atropine* was added to the suppressing mistake.¹⁻⁴ It remained for the bronchoscopist⁵ to revolutionize the whole conception of cough and its relation to the treatment of pulmonary disease. Due credit was given the invisible cilia but it was shown bronchoscopically that the endobronchial secretions, in contradistinction to sputum, are of high viscosity and adhesiveness which clog the cilia and adhere to the walls. It was pointed out that opiates and atropine increase this viscosity and adhesiveness, thus adding greatly to the difficulty of *bechic* expulsion. Due credit was also given to the *bechic* blast, but it was observed that the *bechic* blast could not get under secretions to expel them until they were forced up into the larger bronchi by the *tussive* squeeze.²⁻⁵ It was demonstrated by direct inspection with the eye at the bronchoscope that, in cough, the compression of the lung is one of the most important mechanisms in the natural drainage of the lungs.² A quarter of a century ago one bronchoscopist wrote "The chief function of the cough reflex is to protect the lungs from a dangerous external intruder or internal enemy. As the author has often stated, 'The cough reflex is the watch dog of the lungs.' In view of this function it seems strange that medical men and surgeons continually drug this watch-dog asleep when his efforts are most needed."³ Yet one of the greatest medical men of all time died in the twentieth century, of acute pulmonary infection praising opiates as a medicament for relief of his distressing cough.² In considering the etiology and pathology of bronchial obstruction it is necessary to remember that, as concerns *tussive* expulsion, such obstruction is a relative term. It is always relative to the expulsive power of the *bechic* mechanism of the particular patient at

the particular time. Hence anything that weakens the patient, relatively increases the degree of obstruction. That is why so called "pneumonia" and "hypostatic congestion" (which favors mucosal edema) are terminal phases of so many different diseases.³ With the weakening of the expulsive power of the tussive squeeze and the bechic blast observable objectively with the bronchoscope there probably goes a weakening of the ciliary power, but this is inferential. Bronchial obstruction is favored by stagnation.³ This is not only because of the accumulation. The bronchoscope has demonstrated that prolonged contact of the bronchoscope itself or of any foreign body produces tolerance with sub

operative and postoperative bronchoscopic aspiration.^{3 5 7 8} Probably not more than one in a hundred were really "pneumonia" as the internist uses the term. Many factors may be concerned in bronchial obstruction during general anesthesia. To begin with premedication and the general anesthetic, both, are paralyzers of natural peroral drainage.^{2 3 5} To make matters worse the universal postoperative use of morphine and atropine prolongs the bad effects of premedication. Prolonged operation proloongs stagnation and mucosal tolerance,⁶ and it leaves the patient with a feeble, infrequent cough. Vomiting, whether due to the anesthetic, the premedication, the disease, or the

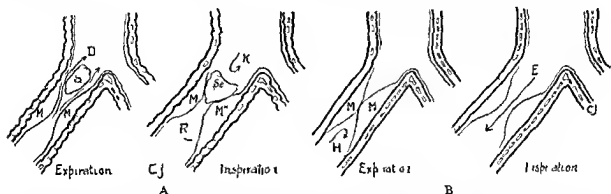


Fig. 465—A. Mechanism of obstructive atelectasis produced by a small mass of secretion (S) acting like a check valve in a pump. It rests in a valve seat of swollen bronchial mucosa at a bronchial orifice (M, M), but at each expiration it is lifted by the expiratory air current, allowing air to escape, as indicated by the darts (D). At the beginning of inspiration the suction pulls the mass (Se) down tightly in the valve seat, so that no air can enter (K). Repeated twenty odd times per minute atelectasis results from pumping air out of the tributary area. A mass of secretion (R) below the inflammatory narrowing (M, M) would have the reverse effect of pumping air in, producing emphysema. Both these valvular mechanisms are seen every day in a busy bronchoscopic clinic. The resulting atelectasis or emphysema has been confirmed by physical signs and fluoroscopy.

B. The expansile check valve, which is the commonest form of valvular obstruction of the bronchi. The swollen mucosa (M, M) comes in contact at the start of the expiratory phase, preventing exit of air, but on inspiration, the enlargement of the bronchial diameter is sufficient to make a small opening for admission of air (E). Promptly at the beginning of the following expiratory phase the diminution of bronchial diameter promptly closes the narrowed lumen, trapping the air below the obstruction. Obstructive emphysema of the tributary area is the result.

sidence of the cough reflex.⁶ Presumably this is due to fatigue of afferent sensory nerve endings, but be this as it may there is no question as to the clinical fact observed at thousands of bronchoscopies. Stagnation is favored by posture, pain on coughing, trauma to the thoracic cage or abdomen, fatigue, old age, feebleness, toxemia, cachexia, shock, drugs, inspiration of vomitus or other kinds of foreign body, and especially by anesthesia, lack of preoperative aspiration, and other factors associated with surgical operations.^{3 5 7 8} Unquestionably most of the dreaded "postoperative pneumonias" were really cases of bronchial obstruction that could have been prevented by pre-

operative manipulations, often introduces obstructive foreign material into the tracheo-bronchial tree.⁸ Blood may be inspired or escape from bronchial vessels, clots are obstructive. Injudicious bronchoscopic procedures are causative in some cases. Patients have come to the clinic with obstruction, some of them with total obliteration of lumen, following endobronchial application of argentic nitrate.

Pathologic Mechanism—The bronchoscope has revolutionized the conception of the pathologic mechanism of bronchial obstruction. Prior to the advent of bronchoscopy, autoptic findings showed bronchi occluded by pathologic

changes in tissues, or by the products of such changes, and it was realized that absorption of air by the circulation could cause atelectasis in the tributary area, but looking into the living moving bronchi revealed the fact that bronchial obstruction in the living is valvular and that the autaptic type of occlusion is only one of four types of obstruction.⁹ The discovery of the valvular mechanism of obstruction threw new light on the interpretation of physical signs. These signs remain, of course, fundamental to all diagnosis of diseases of the chest, especially when supplemented by roentgen ray examination, but bronchoscopic examination is an addition to, not a substitute for, any other method of diagnosis.

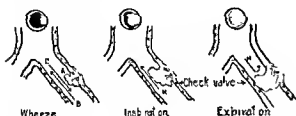


Fig 466—Mechanism of valvular production of obstructive emphysema in the left lung of a woman aged thirty-eight years. A carcinoma (A) on the outer wall of the left bronchus caused a wheeze heard at the open mouth as air passed in and out. When the growth grew larger, inspiratory expansion of the bronchus produced a by passage (M) for air to enter, but at the very beginning of the expiratory phase the diminution of bronchial diameter obliterated the by passage so no air escaped. All three phases were seen bronchoscopically. The result of the repeated pumping in of air was an obstructive emphysema of the left lung. Such cases are common observations at a bronchoscopic clinic. Sometimes we see such a flapping tumor produce emphysema in one segment and at the same time an atelectasis in another segment.⁹

VALVULAR OBSTRUCTIONS OF THE BRONCHI—Valvular obstructions are dependent upon physiologic movements that cease at death, therefore they are not in evidence at autopsy. The valvular mechanisms are not based on theory or inference, they are purely objective findings seen through the bronchoscope by the hundreds annually in any busy bronchoscopic clinic.⁹

The first observation of valvular obstruction was made by the author nearly thirty years ago in foreign body cases. Soon thereafter it was demonstrated that endogenous foreign bodies in form of pathologic tissues and exudates commonly do the same thing. The mechanism in the various pathologic states is illustrated in

Figures 465 to 467. Inspired foreign body is elsewhere herein considered. *Endogenous foreign body* may be in the form of (1) secretion of the high viscosity that has been bronchoscopically

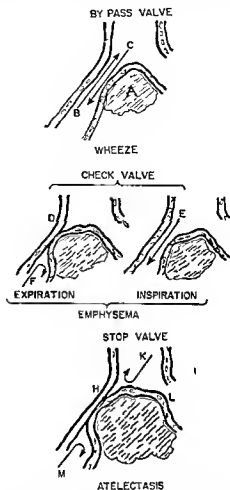


Fig 467—Schematic sketch showing how a peribronchial lesion, tuberculous adenopathy in this case (A), produces, during many months of progress, three different types of bronchial obstruction. From above downward is shown (B, C) first a wheeze produced by compression that still permits respiratory passage of air both in and out. Some months later increase in the size of the mass increases the stenosis so that air can get down to the tributary area during the inspiratory enlargement of lumen, but cannot escape because of the expiratory diminution. The trapped air produces emphysema. Months later still further increase in the size of the mass occludes the bronchus completely by compression, so that air cannot pass in or out and atelectasis follows, as shown in the lowermost illustration. The atelectasis was not accompanied by pneumothorax. The successive stages were observed bronchoscopically.

discovered to be the initial characteristic of all inflammatory endobronchial exudates (Fig 465), (2) pseudomembranous exudates—diphtheritic or fibrinous, (3) sloughs, (4) sequestra

of cartilage, (5) detached fragments of tumor tissue, (6) adenopathy—anthracotic, silicotic, tuberculous, syphilitic, mycotic, or malignant. *Penetrating foreign body* such as a bullet may produce bronchial obstruction either by entering the lumen or by crowding in of the bronchial wall.¹⁰ The same is true of a shell fragment. *Diseases of the bronchial wall* may be mucosal, submucosal, perichondritic, necrotic, tuberculous, syphilitic, or such lesions may have healed and left a cicatricial stenosis. *Tumors* causing bronchial obstruction may be endobronchial, mural (Fig. 466), or peribronchial in location and, of course, benign, borderline, or malignant in character. These are given separate consideration. Bronchial obstruction often occurs in the form of a compression stenosis.¹¹ This may produce obstructive emphysema as shown in Figure 467. Any peribronchial mass can cause compression. Thymic deaths were for years thought to be due to various hypothetical agencies, such as hyperthymization of the blood, until it was discovered at bronchoscopy that *thymic compression stenosis* can mechanically cause asphyxia.¹² The bronchoscopic appearances of various obstructive lesions in the bronchi are shown in Figure 476. The valvular action of a tumor is shown in Figure 468.

Symptoms—Symptoms of bronchial obstruction vary widely. The condition may be symptomless. One lung may be entirely atelectatic yet the patient may not complain of shortness of breath, unless he exerts himself. If the bronchial tree of the other side should become obstructed dyspnea will be increased and asphyxia will be impending. Wheezing heard at the open mouth is always present in partial obstruction of the trachea or a large bronchus (Figs. 465-467). Cough is usually due to inflammatory or other tissue changes and their pathologic secretions rather than to the mere obstruction of a bronchus. The indrawing at the suprasternal notch, that is so conspicuous a symptom of obstructive laryngeal dyspnea (*qv.*), is usually absent in bronchial obstruction. Infective or toxic diseases, of course, add their symptoms to these purely mechanical manifestations.

Diagnosis—That there is an atelectasis or an emphysema or a drowned lung present can usually be determined by physical signs and by fluoroscopic examination, but the character of the obstruction can be determined definitely and conclusively by bronchoscopy and, when ever necessary, by bronchoscopic biopsy. More-

over, the bronchoscope contributes a localization of utmost accuracy by supplying exact information as to conditions at the bronchial orifice of the particular pulmonary segment affected. In addition to biopsy, which concerns tissue only, the bronchoscope has added enormously to bronchopulmonary diagnosis by the accuracy with which uncontaminated specimens of pathologic exudate may be removed from the affected area of the tracheobronchial tree, and from the particular named pulmonary segment, for laboratory study. The field of

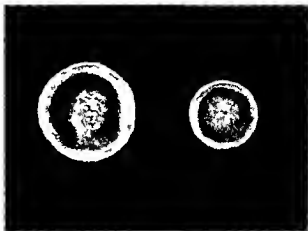


Fig. 468.—Endobronchial valvular action. Bronchoscopic view showing the appearance of an endobronchial tumor (adenoma) and illustrating the mechanism by which it caused obstructive emphysema of the right lung of a girl aged sixteen years. At the left is shown the opening of a passageway for air by the normal inspiratory enlargement of the bronchial diameter. At the start of expiration the normal diminution of bronchial diameter closes the by passage space (right). The air below is thus trapped. Repetition at each respiratory cycle, twenty-odd times per minute, results in obstructive emphysema. This mechanism was first discovered at bronchoscopy and is a common observation at a bronchoscopic clinic. It is never seen at autopsy because it requires respiratory movement. The autopsic position is only the expiratory one shown at the right.

clinical usefulness as well as laboratory research is enormous. To enumerate the conditions would amount to an enumeration of all the diseases of the intrathoracic viscera—in other words, all diseases of the bronchi and lungs as well as compression by diseased neighboring viscera.

Prophylaxis—This is clearly indicated by the etiologic factors, but a few especially important precautions to be taken after operations may be summarized. If there has been vomiting, bronchoscopic inspection and synergistic aspiration are indicated, or, at least, direct laryngoscopic syn-

ergistic aspiration (Figs 469, 470, 471) of the trachea and main bronchi, with an active cough reflex, should not be omitted. If there has been

coughing out of secretions easiest. The upright posture is the most difficult for spontaneous peroral drainage, it is against gravity.¹⁴ What-

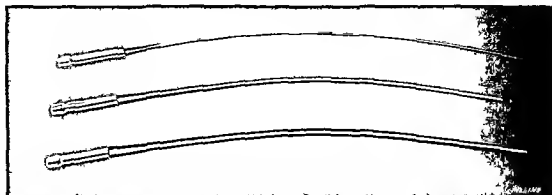


Fig 469 —Silk woven aspiration insufflation tubes for use through the direct laryngoscope. It is vitally necessary to have a free channel around the tube for either aspiration of secretions or insufflation of oxygen. To use the adult size on a baby would be fatal. The lung must not be ballooned by positive pressure. The exact pattern of the distal end is important. There are two eyes with sunken rounded edges at the sides, none in the end. The end is blunt without olive or projection.

no vomiting the catheter aspiration is sufficient, even clots may be thus removed.^{13, 14} These procedures can be easily and quickly done by the anesthesiologist, with the patient on the

ever the posture, it should be changed every hour or two, prolonged maintenance of any posture is unfavorable from the viewpoint of prophylaxis of bronchial obstruction, though

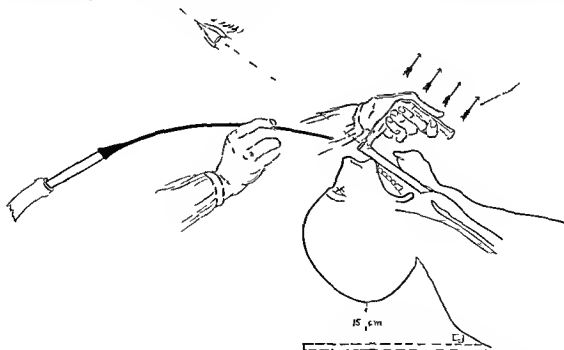


Fig 470 —Exposure of the larynx for synergistic aspiration of the tracheobronchial tree with the silk woven aspirating tube used through the laryngoscope (cf Figs 342, 343). The hands are drawn ungloved for clearness.

ward carriage. When the patient is put in bed, unless there is a special contraindication, the best posture is on the side, without a pillow, the mouth low. This is the posture that renders

there may be, in some cases, contraindications that justify taking the risk. Each time the posture is changed the patient should be encouraged to cough and expectorate. The patient usually

is relaxed, drowsy, and disinclined to cough, especially if the usual postoperative sedation has been given. This medication from the viewpoint of pulmonary complications should be omitted or minimized.^{2 3 5 7} It certainly should not be routinely given in fixed dosage. In cases in which cough is painful, the dosage should be determined with utmost precision to ease the pain of coughing yet not to paralyze the tussive reflex. At least once a day the chest should be gone over to see if there are any areas in which physical signs of atelectasis or emphysema are present. If they are, aspiration (Figs 469, 470, 471) should be done at once. If they do not disappear bronchoscopy is indicated.

ment using the bronchoscope as a speculum. Inspired foreign body can be thus removed in about 98 per cent of the cases. Endogenous foreign bodies can be removed in about the same percentage. The forward grasping forceps for hard substances and the ball forceps for soft substances, like broken down nodes, are best. The distressing dyspnea of asthma (not the disease itself) is due in many cases to the anoxemia of bronchial obstruction by secretion of high viscosity. Relief is immediately afforded in practically all such cases by removal of this secretion. In suppurative conditions, fluid exudative obstructions—even those of the highest viscosity and adhesiveness—are readily re-

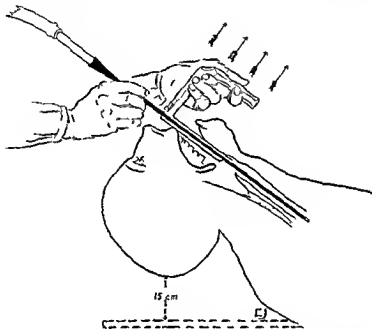


Fig. 471.—Synergetic aspiration of the tracheobronchial tree through the direct laryngoscope. The silk woven aspirating tube is not inserted quite to the carina. The tussive squeeze forces secretions or exudates up into the trachea from any one or more lobes or segments. Synergetic action between the cough reflex and the aspirating tube obviates all necessity for insertion of an aspirating tube into an abscess cavity or a branch bronchus.

Treatment.—The treatment of bronchial obstruction is based upon the accurate determination of the pathologic conditions by bronchoscopic examination of the lesion and the laboratory examination of specimens of tissue and uncontaminated specimens of secretions, exudates, or other material bronchoscopically removed with precision from the diseased area in the particular case. The conditions thus brought by accurate diagnosis within reach of scientific treatment range all the way from the mycoses curable by peroral administration of potassium iodide to cancer calling for thoracotomy. All of the conditions require medical care and management. Some of the conditions require treat-

ment by synergistic bronchoscopic aspiration, which means aspiration in cooperation with the tussive squeeze. As mentioned in a foregoing paragraph on etiology and pathology, bronchoscopic observations show that it is not the bechic air blast that drains the periphery of the lung.³ The compression of the lung during cough squeezes the secretions and exudates from the parenchyma into the larger bronchi.⁵ In synergistic bronchoscopy the bronchoscopic aspirator brings out the pus each time it is expelled from the periphery into the larger bronchi by the tussive squeeze and before it can be inspired by the deep inspiration that follows each cough.^{2 5 8} No attempt is made to insert

the aspirating tube into small branch bronchi, there is no need of doing so, and all risk of even slight trauma from suction of the mucosa into the ootraumatising inlet of the aspirating tube (Fig 469) is avoided. The same principle of synergy applies to aspiration with the silk-woven tube inserted through the laryngoscope (Fig 470). Obviously no general anesthetic is used, and if any local anesthetic or premedication he used it must not be sufficient to inhibit the cough reflex. No local anesthetic is used below the larynx. In cases in which a tracheostomic cannula is worn, such as a laryngectomy or an acute laryngotracheobronchitis, catheter aspiration is done every half hour or oftener by the nurse.¹ For the nurse's use an ordinary soft rubber catheter is best, only the inner cannula being removed. In all catheter aspiration there should never be any negative pressure during its insertion.^{3 4 5}

The treatment of bronchial obstruction caused by *cicatricial stenosis* requires conservatism.^{8 15} In cases of cicatricial stricture due to tuberculosis there is a risk of reactivation if treated actively. All other forms of cicatricial stenosis usually call for treatment if the narrowing is sufficient to interfere with ventilation and drainage of the tributary area. Endoscopic bronchotomy is mechanically easy to do but is dangerous. Bronchial intubation is also easy and is slightly less dangerous. The safest and most satisfactory method is progressive dilatational treatment with the dilators shown in Figure 449. The best of these are the single short olivary dilators. They are of such gradually increasing size that they are quite safe in careful hands. If there are recurrences, as may follow any plan of treatment, a second series of treatments is easily given. Anesthesia is not necessary as the bronchial stricture is not sensitive. The methods of local anesthesia for bronchoscopy in case of adults and of working with out anesthesia in children, are discussed under technique of the endoscopic procedure. *Compression stenosis* anywhere in the tracheobronchial tree yields to the same form of dilatational treatment as advised for cicatricial stenosis. In *aneurysm*, however, any form of local treatment is contraindicated for obvious reasons. Tracheotomy and the wearing of a long enough cannula may be necessary for impending asphyxia. Erosion through by the cannula may be obviated for a time, at least, by using a cannula of the smallest lumen that will give

enough air for all ordinary purposes. *Thymic compression stenosis* is best treated by roentgen irradiation of the thymus gland, pending shrinkage, tracheotomy and the wearing of a long cane shaped cannula is sometimes necessary. Roentgen ray treatment has rendered thymectomy obsolete.¹² Status thymolympaticus chiefly based on theoretic considerations has been attributed to an allergic reaction similar to anaphylactic shock. Such cases may occur, but if so they are a separate entity from the cases seen in relatively large numbers at the bronchoscopic clinic. In these cases children have been rescued from asphyxia by bronchoscopic diagnosis, temporary respiratory relief by tracheotomy, and cure of thymic compressive tracheal stenosis by irradiation.⁴ Our experience at the bronchoscopic clinic indicates that a large area of *congenital atelectasis* may be present without symptoms for a long time, but eventually it will make trouble. Normally inflation of the lungs of the newborn begins with the first cry, but the entire lung is not at once inflated, ordinarily under normal conditions the rudimentary lung is not completely expanded before the third day. Every infant should have careful examination by physical signs and the roentgen ray before discharge from the maternity ward. If any atelectatic areas are detected, a bronchoscopic examination of the bronchus of the affected segment should be made. If the atelectasis is due to obstruction it can be cured by bronchoscopic means. In a number of such cases we have removed a tight plug of semisolid exudate. Congenital web yields quickly to bronchoscopic dilatation with silk woven bougies.⁴ Roentgen-ray examination of the lungs of infants who have had difficulty in establishing respiration should always be made before the child is discharged by the obstetrician. The lungs should be fully inflated on the third or fourth day in normal infants.

Prognosis and Sequelae.—Unquestionably, with the exception of pure-culture infection with aerobic organisms such as mycobacterium tuberculosis, bronchial obstruction lowers the defensive power of the lung. In a general way the prognosis may be said to be good in all forms of bronchial obstruction not due to malignant disease. Bronchoscopic aspiration or treatment in gentle careful hands is practically free from risk. Recurrences may require another series of treatments but the absence of

mortality from bronchoscopy makes this a relatively trivial matter. If long continued, cicatricial stenosis, bronchiectasis, or empyema, or all three, may be regarded as probable sequelae.

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BRONCHOSCOPY IN RELATION TO CHRONIC BRONCHITIS

The term "chronic bronchitis" has been loosely applied at some period in the course of the ailment of every patient with a chronic productive cough, in the absence of sputumary, physical, or roentgen-ray signs of tuberculous or other pulmonary disease. In many of these cases this diagnosis by exclusion was correct, but the bronchoscope, by contributing accurate knowledge of pathologic conditions in the living bronchi of the particular patient, without waiting for autopsy, has shown that in many of these cases there is a localized morbid process that is a very different thing from the diffuse mucosal inflammation that is commonly implied in the term "chronic bronchitis." For example, of 92 consecutive patients referred to the Bronchoscopic Clinic at Temple University Hospital with a working diagnosis of chronic bronchitis forty had localized bronchial stenosis, twenty-one had early bronchiectasis, four had tumors—benign, borderline or malignant, seven were tuberculous, only twenty had nothing more than an objectively evident diffuse mucosal bronchitis. To prevent misunderstanding it should be added that in none of these cases was the bronchoscopic examination relied upon alone. Every diagnostic means known to modern medical science was brought to bear upon every case. For example, incipient bronchiectasis was not considered as excluded without bronchographic corroboration, nor was tuberculosis excluded without the corroborative

opinion of the phthysiologist. The point is that any patient with symptoms of a chronic bronchitis cannot be considered as completely examined unless an objective examination of his tracheobronchial tree has been made with the bronchoscope. This includes, of course, laboratory study of bronchoscopically obtained uncontaminated specimens of secretions, exudates, and granulomatous or other material. The bronchoscopic finding of bronchial obstruction in cases in which no evidences of such a lesion were obtainable by physical signs or roentgen-ray examination is of particular importance, though it does not in any way lessen the value of these fundamentally essential diagnostic means. It implies merely that the finding of no abnormality by these means does not lessen the necessity for bronchoscopic examination when there is productive cough, even though slight. It may be added that bronchoscopic discovery of otherwise obscure localized obstructive conditions is of utmost importance in prophylaxis, as of *bronchiectasis* (*q v*). Unquestionably such localized obstructions, if allowed to progress until revealed by the development of the evidence of roentgen ray and physical signs, would have initiated a self-perpetuating, extending, and incurable bronchiectasis. The class of obstructions referred to are curable by *bronchoscopic treatment*. The appearances and methods are given in connection with bronchial obstruction.

A conspicuous feature of the *bronchoscopic appearances* of a diffuse chronic bronchitis is the altered character of the secretions. Instead of the diffused transparent moisture that normally covers the entire mucosa with a uniform invisible film, the secretions are adherent in patches, tiny plaques, or droplets that are more or less opaque with whitish or yellow tinged cloudiness, sometimes greenish. If sufficient in quantity, they may show passive wafting in the air current. The mucosa early is thickened, reddish, and velvety, in older stages the mucosa is cicatricial, patchy, and streaked with branching vessels.

Having excluded bronchial obstruction, and having corroborated a diagnosis of chronic bronchitis without obstruction, bronchoscopic methods of local treatment are not alone sufficient for cure, they are of aid to medical care and management. Throughout the treatment of such cases it must always be borne in mind that

chronic mucosal inflammation always involves a potential of obstruction by the luminal encroachment of inflammatory thickening of walls, impairment of ciliary action, and by stagnation of exudative products difficult to expel. Inspection and aspiration are indicated at frequent intervals, usually at least weekly. Endobronchial medication may be used as indicated but in most cases there is nothing better than an endobronchial spray of sterile normal salt solution which greatly facilitates not only bronchoscopic aspiration but renders the secretions more manageable by the cilia. If the secretions are especially viscid the salt solution should have an addition of 15 grains of sodium bicarbonate (1 gm.) to 1 ounce (30 cc.) of the solution. This will usually lessen cough, not by the pernicious suppression of sedatives but by cleansing the tracheobronchial mucosa. In fact this promotion of ciliary drainage¹ is subjectively so comforting that the patient is prone to rely solely on it to the neglect of strict adherence to the regimen of rest and medical care and management which are essential for cure (see under "Bronchiectasis," also "Bronchoscopy in Relation to Nontuberculous Pulmonary Abscess"). The relative humidity of inspired air throughout the twenty-four hours should never be lower than 65 per cent, 75 per cent would be better. All opiates, atropine, and all sedative "cough mixtures" must be absolutely forbidden.

The prognosis as to cure is good for patients who will cooperate in the treatment and regimen of general medical care and management. There may be recurrences after acute infections of the respiratory tract but with prompt attention these reinfections will not drift into a chronic condition.

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BRONCHOSCOPY IN RELATION TO ASTHMA

Broadly speaking modern thought is that the manifestations of asthma, being largely and invariably concerned with the tracheobronchial tree, render it advisable in every case to look into the tracheobronchial tree. This of course does not in the slightest degree imply that a bronchoscopist should undertake alone the diagnosis or the treatment of asthma. Bronchoscopy is an addition to, not a substitute for, other methods of diagnosis, treatment, or research in this problematic disease. In any case of asthma "the allergist should be at the helm."¹ For the purpose of consideration of the relation of bronchoscopy to the problem of asthma it may be stated that four classes of cases are referred to the bronchoscopic clinic (1) allergic, (2) infective, (3) combined allergic and infective, and (4) those in which there are some of the clinical features, chiefly symptomatic, of asthma but no true asthma as the term is now restricted. Our experience is that allergists now recognize the importance of completing diagnostic studies by adding a seriatim examination of the orifices of every segmental branch as well as of the entire tracheobronchial tree in cases of asthma even those of purely the allergic type. In the infective and the combined types the bronchoscope is of aid in supplying not only information as to pathologic conditions present in the particular case, but also in supplying uncontaminated specimens of bronchial exudates for three purposes, namely, (1) studies, (2) investigation of the possibility of bacterial sensitization in the particular case, (3) preparation of autogenous vaccines, if desired. In the fourth and largest class of cases we have found all varieties of bronchial obstruction (*qv*). Many of these patients had typical positive reactions to sensitization tests as a cocurrent phase, which rendered accurate knowledge of the existing endobronchial pathologic conditions essential for the solution of the diagnostic, etiologic, and therapeutic problems.

Confusion has arisen in many of the cases from causes explained by certain clinical facts relative to dyspnea often observed at the bronchoscopic clinic (1) Dyspnea of any kind is usually worse at night (2) Nocturnal dyspneic attacks are suddenly precipitated by the occlusion of narrowed bronchial lumina by se-

cretions or exudates accumulated during the first few hours of sleep (3) Nocturnal attacks are often precipitated, even in normal persons, by the overflow into the larynx of secretions accumulated in the pharynx during a period of sleep (4) Sudden air hunger causes panic, violent attempts at inspiration, and the more violent and sudden the attempts are the less air passes the larynx (Fig 357) Such attacks are sometimes initiated, as just mentioned, by the paroxysms of cough and panic following the overflow into the larynx of secretions accumulated in the pharynx. It may be regarded as axiomatic that in all cases of any kind of dyspnea differential diagnosis is often misleading, objective examination of the laryngotracheobronchial airway is essential. The bronchoscope supplies the means. In many of the cases in class 4 there was not dyspnea in the sense of difficult or labored breathing, nor any air hunger, but only a wheezing sound. Of course the alert practitioner of today recognizes the truth of the aphorism "all is not asthma that wheezes",² and it is now recognized that bronchoscopy is indicated in all cases in which there is wheezy respiration, whether constant or paroxysmal, nocturnal or diurnal, negative or positive to allergic tests. Typical slow expiratory wheezing respiration occurs in a great variety of tracheobronchial conditions ranging from the presence of peanuts in the trachea or bronchi in children to cancer in adults.

The foregoing refers to diagnosis. In the treatment of asthma, *bronchoscopic aspiration* in a large proportion of cases affords dramatic benefit.³ In most cases of bronchial asthma there is thick, adherent, viscid secretion which contributes largely to the bronchial obstruction, hence to the patient's air hunger. Aspiration of this secretion gives relief of distress in many cases that seems little short of marvelous. In some extremely severe cases it has prevented asphyxial death. Except for an occasional diagnostic examination, the bronchoscope need not be used, the aspiration can be done with the silk-woven aspirating tube (Figs 468, 469, 470) passed through the laryngoscope. The frothy character of the sputum in asthma is due to small bubbles caused by pumping air through the tenacious sticky secretion. This process is visible at bronchoscopy. The prognosis as to relief of distressing symptoms is greatly improved by the addition of bronchoscopy to

remedial measures. The prognosis as to cure usually remains a problem of allergy and internal medicine.

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BRONCHOSCOPIC TREATMENT OF ASTHMATIC CHILDREN

Bronchoscopy in the asthmatic child does not differ in its purpose from bronchoscopy in the asthmatic adult. This purpose is (1) inspection of the tracheobronchial tree, (2) removal of secretion, and (3) preparation of vaccine.

Direct examination of the bronchi may reveal a foreign body as the cause of an asthmatic wheeze, even in the patient who reacts positively to skin sensitization tests. Inspection will also determine the presence of changes in the tracheobronchial membrane, in repeated bronchoscopic examination of 100 allergic children, ninety-one showed nonspecific inflammatory changes. Sixty of the one hundred children had thick mucoid or mucopurulent secretion in the lower airway. The presence of thick secretion is the most constant finding in children bronchoscoped during an asthmatic attack. In instances in which the attack cannot be controlled by medical treatment, bronchoscopy invariably permitted aspiration of viscid secretion and gave respiratory relief of varying duration. As the cough reflex in children is a relatively ineffective mechanism, aspiration of this obstructing secretion is even more necessary in the child than in the adult.

Autogenous vaccine prepared from the bronchoscopically aspirated secretion is a tremendous aid in the therapy of asthma in children. Infection apparently plays an even more important role in the asthmatic child than it does in the asthmatic adult. Since the onset of asthma in the child is frequently coincidental with acute

infections, the use of vaccines is a logical treatment. Vaccine prepared from the bronchoscopically aspirated secretion should be given in doses small enough to cause no general or local reaction. This often means diluting the material to 50,000,000 bacteria per cc or even to 25,000,000 or to 10,000,000 per cc. Even at this concentration only 0.01 cc may be tolerated. The object is to find the dose which will give no reaction and then judiciously increase the amount, keeping always below the reaction level. For optimum effect, the use of the vaccine must be continued for a year after all clinical symptoms have disappeared.

Contraindications.—Bronchoscopy is best not done during acute infections with fever, because of a possibility that instrumentation may increase inflammatory reaction. However, in the patient whose respiration is embarrassed by thick bronchial secretion, there is no contraindication to aspiration.

Technic.—Anesthesia, local or general, is not needed for bronchoscopy in children. Cocaine should not be used because of its relative toxicity for the young. A frank discussion about the procedure, with emphasis on its brevity, will usually gain the patient's cooperation.

The young patient reacts more violently to instrumentation than does the adult, and the allergic individual is always particularly susceptible to swelling of his soft tissues. Therefore, in the allergic child every effort must be used to avoid any possible irritation. That this can be done is evinced by the fact that in over 1500 bronchoscopies done on asthmatic children we have never experienced an alarming postoperative reaction. To achieve this result three factors are necessary: (1) proper size of endoscopic tubes, (2) brevity of procedure, and (3) gentle manipulation. With these factors meticulously observed the child will usually accept bronchoscopy with more philosophy than is exhibited by the adult.

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BRONCHOMYCOSIS

Bronchomycosis is disease of the bronchi due to fungi. Pneumomycosis is a synonym. Various synonyms apply to cases in which the fungi have been identified, as, bronchoaspergillosis, bronchoblastomycosis, bronchomoniliasis, bronchomucomycosis, bronchooidiosis, bronchospitrichosis.

No statistics afford any true indication of incidence because the disease has been rarely diagnosed as a morbid entity. The various diagnoses include many lesions that are present, as mentioned under "Pathology," but the underlying or associated factor of mycotic infection has been overlooked. Now that the bronchoscope has brought precision to bronchopulmonary diagnosis and has developed the new science of bronchology, statistics of bronchomycosis will acquire value. Bronchomoniliasis, from *Monilia albicans* infection, is becoming increasingly frequent.

Etiology.—The rapidly developing department of medicine known as *bronchology* is shedding light on the etiologic problems of mycotic infections of the bronchi. The bronchoscope has substituted precision for the formerly used bewildering examination of the floral profusion of sputum, composed as it is of a mixture of secretions from the nose, nasal accessory sinuses, nasopharynx, pharynx, tonsils, tongue, and mouth, to which are added cultures grown on the favorable culture media afforded by food lodged in carious dental cavities and interdental spaces and to these were added the collections of free fungi filtered out of the inspired air by the turns and moist walls of the upper air passages. Among the fungi that have been bronchoscopically found in lesions of the bronchopulmonary system are blastomycetes, actinomyces, aspergilli, coccidia, sporotricha, mucors, and monilias. Botanical classifications are as yet confused, overlapping, and changing, as to pathogenic fungi. *Nocardia*, a name formerly given to a genus of organisms causing the well-known disease, bronchocardioidosis, is now classified under actinomyces. Of the many varieties of monilia only *Monilia albicans* has been found pathogenic for man. Notwithstanding the fact that practically everyone is frequently exposed to infection by fungi yet relatively few acquire fungus disease. This raises the interesting problem of predisposing causes, lowered resistance, and the possibility of symbiosis and

secondary invasion. Little is definitely known about any of these phases of the subject. Molds have always been suspected as causative of disease, yet some varieties of quite wholesome cheese depend upon mold for flavor and consistency, yeast fungi yield vitally necessary vitamins, and to cap the climax the genus, *Penicillium*, dreaded by housewives, has yielded not only creaminess, flavor, and color to Camembert but also the most powerful non-toxic bacteriostatic drug known to man. An enormous field awaits research into fungus diseases and bronchomycosis is an important part of it.

Pathology.—The route of invasion has not been definitely determined—the oral, the dermal, the lymphatic, the aerial, and the mucosal routes, each has had its share of blame. Whatever the route, the resultant lesion in most of the bronchomycoses is a chronic granuloma resembling a tuberculo-granulomatous process. A destruction of adjacent tissue cells is accompanied by an attraction of leukocytes, then surrounding cells begin to proliferate. Bronchostenosis follows. Connective tissue proliferation may result in mass formation resembling a tumor. Large masses or smaller lesions may break down and become open ulcerogranulomatous ulcers involving the bronchial wall. Abscess, single or multiple, may form. Drowned lung also occurs.

Symptoms.—Cough, at first dry and sometimes remaining so, is always present. As the disease progresses the cough becomes productive. In case of yeast fungi like *Monilia albicans* it may be accompanied by a yeast like odor on the breath. Fever is of an irregular type, usually moderate unless there is an undrained suppurative process, the latter is accompanied by chills, sweats, and weight loss. The clinical features of bronchomycosis closely resemble those of bronchopulmonary tuberculosis.

Bronchoscopic Appearances.—In some cases a concentric ring of infiltration of the wall narrows the lumen almost to the point of obliteration. In other cases the lesion may occupy only part of the periphery. The mucosa is reddish and inflammatory, rarely edematous. Granulomas in some cases may look as if epithelialized. Ulcerogranulomas of the bronchial wall are usually raised, stenotic, and seen on edge. The lesions or the inflammatory wall may be smeared with blood. Segmental bronchial orifices may be blocked by edema, soft granulomas, bleeding

granulations, or they may be so completely obliterated as to be difficult to find. In bronchoactinomycosis the early lesions are small and discrete, later they are large, copiously discharging ulcers, and pus is usually seen emerging from segmental orifices. The secretions in these cases may be so thick as almost to clog the aspirating tube. In other bronchomycoses the secretions may be mucopurulent, purulent, bloody, thick or thin. The odor may be yeasty, moldy, or offensive. In some cases, at certain stages, the secretion may be scanty and not purulent.

Diagnosis—The diagnosis is made by microscopic examination of smears, of cultural growths, and of tissues to identify the fungi. Inoculation of laboratory animals is also important. Uncontaminated specimens taken by bronchoscopic methods render the diagnostic procedures conclusive. They serve perfectly in the differential diagnosis among the various mycoses and also in differentiation from bronchopulmonary tuberculosis, which so closely resembles bronchomycosis in clinical features. Except in actinomycosis, which usually shows the characteristic yellow granules of ray fungus, sputum examination is useless and often misleading for diagnosis. In addition to the confusing uncertainty of sputum examinations, as mentioned under "Etiology," there is the additional fact that in some of the bronchomycoses the characteristic organisms are not abundant and are not readily found in sputum, moreover, when found they are not necessarily from a bronchial lesion. Worse yet, there may be no sputum. The only certain and significant way to find the fungi is by bronchoscopic biopsy. This is especially true of blastomycosis. The blastomycetes are found by microscopic examination of unstained specimens of tissue.¹ In some cases of bronchomycosis, the specimen will be from relatively firm tissue, in others from a pinkish or pale granuloma, and in other cases from a bleeding mass of granulations. Syphilis may coexist, and serologic tests for this disease should never be omitted.

Associated and Complicating Diseases—The disease must frequently associated with bronchomycosis is pulmonary tuberculosis. It may precede or follow the mycotic invasion. Dermatomycosis is a relatively frequent associated lesion, it is usually but, curiously, not always by the same species of fungus. The mouth, tongue,

lips, and anterior nares are quite commonly a primary site of the same infection that is found in the tracheobronchial tree. In any case of fungus infection of mucosal or dermal surfaces, examination of bronchoscopically removed specimens of bronchial secretions should be carefully made. Some of the dermal fungi have never yet been reported as found in the bronchi, but in view of the past suffering of humanity from incomplete medical examinations it seems best for the patient that the physician should not decide, on the basis of theoretic considerations, that any examination is unnecessary. In one of our cases a bronchoblastomycosis was associated with an esophageal blastomycosis, not as an integral lesion, and the patient developed also a pulmonary tuberculosis. All of these lesions were identified by bronchoscopically removed specimens of tissue.² In another case of bronchoblastomycosis the trachea and then the larynx became involved, requiring tracheotomy. The blastomycotic process extended out through the fistula and involved the skin.¹ There was no prior dermal lesion, a reversal of this sequence occurred in another case. Bronchopneumonia is sometimes a complication, usually as a terminal phase. It is often simulated by and mistaken for atelectasis, usually segmental, sometimes lobar, rarely a whole lung may be atelectatic. In bronchoactinomycosis the process may extend through the thoracic wall forming fistulas with dermal involvement at the orifices.

Treatment—In all forms of bronchomycosis except bronchoactinomycosis, potassium iodide is curative.^{1, 2} In bronchoactinomycosis it is helpful in the early stages. It is given in doses of 40 to 60 grains (2.5 to 4.0 gm.) daily, well diluted with water. The treatment must be continued for a long time and notwithstanding any symptoms of iodism that may appear. Copper sulfate has been used, but is not so efficacious, and it is not well tolerated. Local medication is not indicated, but frequent synergistic laryngoscopic aspiration is helpful. General treatment should be in the form of a strict bronchial regimen (q v).

Prognosis—Bronchomycosis must always be considered serious, but in early stages it is curable. Bronchoactinomycosis is the most intractable and the mortality is high, probably over 50 per cent.

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ACUTE INFECTIVE LARYNGOTRACHEOBRONCHITIS

Acute infective laryngotracheobronchitis is an acute inflammation of the upper and lower air passages with specific clinical features but not always attributable to the same infective agent. The name, laryngotracheobronchitis, first given as a title¹ in 1922, though previously used in descriptions,^{2,3} is unwieldy but no synonym has since proven acceptable. Older names that undoubtedly were applied to the same disease, though they were not based on bronchoscopic observations, are *laryngotracheite* (Blaud, 1823), *bronchite suffocante* (Belloc, H, 1837), *laryngotracheite pyogene, pas meningogène* (Trousseau, 1851), *suffocative bronchitis, catarrhal fever, nonmembranous croup, suffocative catarrh, streptococcic bronchitis*. Cases occurring as a phase of epidemic influenza were called *influenza, la grippe, tracheobronchite grippale, laryngite sous glottique grippale*.

One epidemic of laryngotracheobronchitis is referred to in the literature.¹ In most instances authors report a group of cases too few to be regarded even as endemic. However, in view of the fact that most of the cases of this disease are overlooked it might have been epidemic or endemic at the time, but recorded as another disease, bronchopneumonia for example. Even today it is not always easy to convince the practitioner that the disease is a morbid entity. In my experience sporadic cases have occurred every winter from 1889 to 1939. In some years only two or three cases were referred to the Bronchoscopic Clinic, but an increase in number always corresponded to the prevalence of acute infection of the respiratory tract, including so called influenza, when this happened to be the name given to the prevalent infective ailment. Vital statistics afford no criterion whatever for estimating incidence. Not being a reportable disease the only statistics in which

it could be recorded would be those of mortality. In this class acute laryngotracheobronchitis has been officially ignored as a morbid entity, hence all deaths from this disease are recorded under other diagnoses, many of them under "croup," some as diphtheria, probably most of them under the all-covering diagnoses of pneumonia, and bronchopneumonia. Doubtless the latter was correct as to the terminal phase of the disease in many of the cases. Compared to the exanthemas the disease is uncommon, but there is no way of calculating the number of cases of laryngotracheobronchitis that are diagnostically overlooked. Now that the case with which a child's larynx can be examined is being recognized, statistics on incidence of this disease will develop value.

Etiology.—The chief predisposing cause is age. Part of this predisposition is probably the lack of partial immunity in a child who never has had an acute respiratory infection,⁴ but the chief part is anatomic. Loose areolar tissue is abundant in the larynx during the first few years of life and the lumen of all the air passages below the larynx is small. It is natural to assume that a small child needs only a small tube, but, in a passageway, size is absolute, not relative. Every plumber knows that a small pipe is more easily "stopped up" than a large one and all sanitary engineers specify only large pipes for drainage. As a mathematic problem, for example, 1 mm. of exudate lining the wall of a bronchus 2 mm. in diameter will occlude the bronchus, whereas it would leave a 2-mm. lumen in a 4 mm. bronchus. The anatomic is combined with the physiologic fact that the highest air pressure a baby can raise in a bechic blast is not one tenth of that of an adult. Therefore the baby is handicapped by three basic factors: namely, (1) a weak bechic blast and tussive squeeze, with which to expel (2) sticky secretion, through (3) small tubes. To these factors must be added another one of the characteristics of the disease—the toxemia obtunds the cough reflex. Total absence of this reflex occurs in some cases.^{1,2} In the fifty-year period previously mentioned it was frequently noted that, during an epidemic of infection of the respiratory tract, the older members of a family had the usual manifestations while the child under two or three years of age developed this disease we call laryngotracheobronchitis, evidently due to the same infective agent. All of these facts, physical and clinical, establish age

as the basic predisposing factor. In other words, it is not a specific primary or secondary invader but the age of the child that gives the clinical features that constitute a morbid entity. These facts are not inconsistent with the known facts in the bacteriology of the disease. In our experience⁵ hemolytic streptococci predominated—they were often present in pure culture (bronchoscopic specimens)—yet all the clinical features of the disease occurred in some of our cases in which other bacteria (*Streptococcus viridans*, *Staphylococcus haemolyticus*, pneumococci, *Hemophilus influenzae*) predominated or occurred in pure culture. Font and Ortiz⁶ report typical cases of laryngotracheobronchitis in which the predominating organisms were a hemolytic strain of *Staphylococcus aureus*. This is especially striking because streptococcal infections in any part of the body are mild and rare in Puerto Rico. For the present, therefore, we may say that any virulent pathogenic infective agent affecting the air passages may produce the disease in a child under, say, three years of age, we must leave to the future the further study of filtrable agents, primary and secondary invasion, symbiosis, and apparent specificity resulting from quantitative rather than qualitative factors due to capacity of different strains to produce toxins.

Pathology.—One outstanding pathologic factor in the disease, first noted in the living patient with the development of direct methods of examination,^{1 2 3 7} is variously described from its endoscopic appearances and behavior as "thick tenacious exudate" that "infants are unable to expel," "tough," "tenacious," "adherent," "sticky," "gummy," "viscid," "of high viscosity," "forming clots and crusts." This secretion does not often appear clinically because young children do not expectorate and could not get the exudate up if they did. Microscopically this exudate is found to contain mucus, leukocytes, epithelial cells, broken-down debris, and fibrin. The gross appearances of the pathologic tissue changes are described in the following paragraph. Microscopic examination in autopsic cases⁸ shows infiltration of bronchial and alveolar walls, some alveoli are partially collapsed, others contain leukocytes with some fluid. Small bronchi are seen plugged with polymorphonuclear leukocytes. In many locations glandular elements are damaged, few are destroyed.

Direct Examination.—The mucosa of the

larynx is a deep red, slightly less deep on the cords. The subglottic tissues on each side extend medially, showing as mounding, intensely red, semi-elliptic folds, one below each cord (Fig 345a). These swellings, peculiar to children, are due to the loose connective tissue in the *conus elasticus* in childhood. Small patches of secretion may be seen but they can be wiped away cleanly, leaving no eroded or bleeding surface, there is no ulceration, no membrane comparable to that in diphtheria. The living pathologic condition of the bronchi is interesting and important. The progressive appearances were vividly described twenty-two years ago as follows:⁵ "The tracheal mucosa is reddened. Its color deepens. Swelling of the mucosa begins. Later on exudate forms, at first serous, then mucoid, then purulent and finally thick, tenacious and exceedingly difficult of expectoration even by the robust adult. In infants who are naturally almost incapable of expectoration, death may occur from inability to rid the air passages of secretion (drowning of the patient in his own secretions). The bronchi, or even the trachea itself may be occluded by mucosal swelling, or edema, actually causing death by stenosis. Both these conditions are independent of bronchopneumonia, which may or may not exist." Subsequent observations have fully confirmed these observations. As in other conditions the rings, so characteristic of the normal, are obliterated by swelling. Some orifices are noted as filled with pus, and absence of bubbling indicates no passage of air. Still other orifices are found obstructed with straw-colored or brownish crusts. These crusts have the appearance of dried serum with little admixture of pus. It is true that thick pus, too viscid to run down the glass walls of the collector, is seen in various bronchial diseases,⁹ but no such gum-like and crusted secretion as that just described is ordinarily seen in any disease other than the malady under consideration. In rare instances an outstanding feature of the endobronchial picture is the dry, sometimes even glazed, appearance of the tracheobronchial mucosa.⁴

Symptoms.—In most of the cases the initial symptoms are those of "a cold in the head." These symptoms are soon followed by hoarseness, croupy cough, stridorous breathing, air hunger, restlessness, signs of obstructive laryngeal dyspnea, ashy gray pallor, violaceous lips and finger nails, irregular fever, and toxic prostration. The symptoms in the foregoing list

are characteristic though some may be absent in a particular case. In some cases, at later stages even cough may be a minor symptom or absent.^{2 3 4} Fever may be moderate, but usually rises high in the terminal phases of fatal cases.

It will be noted that one of the chief characteristics of the living pathologic condition as shown in the endobronchial picture is the obstruction of the larger bronchial orifices. The other characteristics have an important bearing on the physical signs. The effect of the secretion may be to produce areas of either atelectasis or emphysema, usually both in respective parts of the lung. The atelectatic areas are almost always mistaken for consolidation. An overlying area of emphysema may bring the percussion note up to a normal vesicular or to a vesiculotympanic resonance, but in such cases a muffled quality of the tympany is usually perceptible. The mechanisms producing these pathologic states and their corresponding physical signs are described in connection with bronchial obstruction. Bronchial breathing and bronchophony may be present in atelectasis of larger areas. Breath sounds may be suppressed or absent especially at the bases. In many cases the physical changes are slight to a degree out of all proportion to the endobronchial picture and the general illness of the child. In the dry stage rales are sometimes coarse and they may be heard all over the chest when the secretions are very thick.

Diagnosis.—The laryngoscopic and bronchoscopic appearances given in a preceding paragraph are diagnostic. The appearances in the rare cases of tracheobronchial diphtheria with no membrane in the fauces or pharynx are similar early, but the appearances of diphtheritic membrane in the trachea and bronchi are unmistakably characteristic. Moreover, as between diphtheritic and nondiphtheritic laryngotracheobronchitis, the bacteriologic diagnosis is of primary importance. Carriers of diphtheritic bacilli in the larynx and tracheobronchial tree are, in our experience, rare compared to oral and pharyngeal carriers. In the thousands of specimens of secretions that have been taken from deep in the tracheobronchial tree in the bronchoscopic clinic, we have not found diphtheritic organisms in a patient who did not have a diphtheritic process in the form of active disease. It therefore seems justifiable to assume, for diagnostic purposes, that any child

with the objective appearances described, and repeatedly negative reports from smears and cultures of material obtained bronchoscopically from the interior of the tracheobronchial tree, has an acute infective laryngotracheobronchitis and not diphtheria. These statements refer particularly to cases in which there is no membrane in the fauces.

Prophylaxis.—The disease is infectious but prevention of exposure is practically difficult. Infected air can be sterilized with ultraviolet radiation but this also awaits solution of problems of practical application. Wintering in the torrid zone is prophylactic but is available for few.

Complications.—Streptococcal or other infection of the blood stream is the most frequent complication, next in frequency is cardiac or pericardial disease. Bronchopneumonia is a terminal complication.

Treatment.—Mainly, treatment should be directed toward (1) guarding against (a) asphyxia, (b) dehydration, (c) exhaustion, (d) medication with opiates and atropine, and (2) the combating of infection.

To prevent asphyxia, increase of the oxygen content of the inspired air may be helpful for a time but it must be remembered that a prolonged fight for air may, by long continued anoxia, fatigue of the respiratory centers, and exhaustion of the child, preclude all chance for recovery. Tracheotomy should be done when the cardinal signs of obstructive laryngeal dyspnea are clearly present, and the tracheal incision should always be below the second ring. Intubation (q.v.) may be done if an expert intubator is always to be immediately available in case the tube should become obstructed or coughed out. It has the disadvantage that it prevents drainage, whereas after tracheotomy the nurse can keep the trachea and larger bronchi clear by aspiration with a soft rubber catheter passed through the cannula as often as necessary, as mentioned in a subsequent paragraph. If the child seems to get worse, the ashy pallor increases, the percussion note over the bases becomes increasingly impaired and rises higher in level, and if the catheter aspiration fails to relieve these symptoms, bronchial obstruction, not pneumonia, is most likely the cause. A small, short bronchoscope should be passed through the tracheotomic orifice. Almost always these signs will be found to be due to a plug, clot, or crust blocking one or more of the

larger bronchial orifices. Removal with bronchoscopic aspirator, or by forceps, if necessary, will give immediate relief and a remarkable change for the better in the patient's condition. The obstruction may be unilateral or bilateral, and may recur a number of times each time the same good result is usually obtainable.

The use of the bronchoscope and the forceps can be minimized in frequency and in some cases rendered unnecessary by early and careful guarding against dehydration. There are two phases to this important part of the treatment, drying of the secretions and systemic dehydration of the patient. In winter the indoor air in this country is extremely desiccating. To prevent it drying the bronchial secretions into plugs and crusts the inspired air should have its humidity percentage increased close to the dew point, which is 100 per cent, without raising the temperature over 70°F. This is best done with a mechanical humidifier in the room. If there are signs of anoxemia the oxygen percentage may require increase also. If properly managed, an oxygen tent can supply these conditions. The dehydration of secretions in tracheotomized patients can to a great extent be prevented by supplementing atmospheric humidity by instillation of warm sterile salt solution by medicine dropper into the trachea. The frequency with which this is to be repeated will depend upon the percentage of humidity of the inspired air and the character of the secretions in the particular case. In some cases it may be required every hour or two. As a rule it should be soon followed by tracheobronchial aspiration with the sterile soft rubber catheter. Repetition of both of these procedures may well be left to the judgment of a nurse experienced in such cases. The facility with which the airway can be kept clear is one of the advantages of tracheotomy over intubation, in which even the physician is helpless in removal of secretions without removal of the tube. The long struggle with obstructive secretions in laryngotracheobronchitis is quite different from the short period of waiting for the effect of antitoxin in tracheobronchial diphtheria. If chemotherapy prove as efficient for pyogenic infections in this region as antitoxin for diphtheria, intubation might be preferable in many cases. The drying and increase in viscosity of secretions can be much lessened by avoiding use of opiates and atropine. These drugs also block drainage by paralyzing the

cough reflex. Their injurious and often fatal effect in such cases has been pointed out frequently for many years.^{1 4 9 10 13} The prevention of systemic dehydration of the patient is assured by attention to intake of water from the moment the presence of laryngotracheobronchitis is suspected. The nurse should encourage the child to drink and the water should be cool and palatable. When the patient becomes too toxic to drink the offered water, the stipulated total amount should be given by dropper in the mouth. Unless comatose the patient will swallow it, especially if cool. Stipulation of the amount is essential for certainty. Hypodermoclysis, also intraperitoneal and other uncomfortable and disturbing methods of administration of fluids, can and should be thus obviated, or, at worst, postponed. This has an important bearing on the avoidance of exhaustion.

The disease is self limited. One of the greatest dangers in the disease is that the child's strength will not last until the long course is run. Every thing must be done to minimize exhaustion. The child's rest should not be disturbed except for essentials and these should be at longest advisable intervals. This applies to nurse's attention as well as physician's examination. Transfusions are excellent but even this minor annoyance can be made less frequent by proper conservation of strength as just mentioned. The room should be quiet but not darkened, the child should have all possible sunlight and day light. All of the just mentioned matters by increasing resistance go a long way in the combat against infection.

In recent years powerful remedies have been added to our means of bacteriostasis by chemotherapy. The high mortality rate of laryngotracheobronchitis fully justifies whatever toxic risks there may be in the use of sulfathiazole and other sulfonamides. These toxic risks how to minimize them the dosage, and signs rendering an immediate stopping of the drugs, receive the extensive consideration they require in the section "Chemotherapy in Otolaryngology". The use of the even more promising nontoxic penicillin is also given there.

Course.—Laryngotracheobronchitis is a self-limited disease, running its course in about two weeks, though complications may extend it to four weeks. Chemotherapy may shorten it.

Prognosis.—Prior to the advent of chemotherapy the mortality was about 45 per cent in

children under two years of age. In children above this age the rate was progressively less until at adolescence the disease disappears as a morbid entity, being replaced by infective tracheobronchitis as seen in adults. Cardiac complications and blood stream infections usually have been fatal. The cases so far treated by chemotherapy are not yet so numerous as to afford significant data on the mortality rate. The outlook is greatly improved.

Sequelae.—Pulmonary abscess, bronchiectasis, valvular heart disease, and laryngeal as well as bronchial stenosis have been reported. Difficult decannulation is a sequela that is avoidable by doing the tracheotomy below the second ring.

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HEMOPTYSIS

Bronchoscopic Diagnosis.—The appearance of blood, whether as streaks in clear mucus or as pink pus or as mouthfuls of blood, always presents a problem calling for immediate search for its source. Assuming that the mouth, gums, nose, nasopharynx, pharynx, esophagus, and larynx are proved not to be the source, bronchoscopy is urgently indicated. In our clinic this is a common indication for diagnostic bronchoscopy. We have found that, contrary to the general opinion, pulmonary tuberculosis is not the commonest cause of hemoptysis. A search of our records revealed 446 cases of non-tuberculous hemoptysis, in which bronchoscopic search was made for the source of bleeding. The bronchoscopic diagnoses were made by appearances and by laboratory examination of bronchoscopically removed specimens of secretions, exudates, and, in some cases, tissues. The results in this series were as follows:

Bronchiectasis	138
Primary carcinoma of bronchus	82
Tracheobronchitis	74
Pulmonary abscess	51
No evidence of disease	34
Nonsuppurative pneumonitis	15
Suppurative pneumonitis	11
Adenoma of bronchus	11
Secondary cancer of lung	6
Lobar atelectasis	4
Primary carcinoma of trachea	2
Suppurating pneumoconiotic lymph node discharging into bronchus (anthracosis)	1
Nonspecific granuloma of bronchus	1
Streptothricosis	1
Chondroma of bronchus	1
Osteoma of trachea	1
Dermoid cyst communicating with bronchus	1
Bronchiolitis	1
Neurofibroma involving wall of bronchus	1
Total	436

In cases of copious hemorrhage it may be best to postpone the diagnostic bronchoscopy for a few days. On the other hand, there may be cases in which bronchoscopic hemostasis is indicated for severe bronchial hemorrhage. Tethered gauze plugs may be used for this.² In one of our cases, in an almost exsanguinated patient with bronchial adenoma, the hemorrhage was arrested by bronchoscopic insufflation of bismuth subcarbonate in dry powder.³

Bronchoscopic Aspiration of Blood and Clots.—In all cases of blood—fluid or clotted—in the tracheobronchial tree, regardless of how it got there, removal is desirable. Blood is a good culture medium and bronchoscopic aspiration is a good and, in fact, the only known means of getting it out when not expelled. This aspiration is a powerful factor in the *prophylaxis of bronchial obstruction, bronchiectasis, drowned lung, and pulmonary abscess*. The aspiration of blood and clots is indicated in *injuries of the chest wall or lung*. In all of these cases premedication or anesthesia must not be sufficient to lessen cough during aspiration. The bronchoscopist needs the aid of the tussive squeeze⁴ to force blood and secondary materials up into the larger bronchi for ready aspiration (see "Synergistic Bronchoscopic Aspiration of Pulmonary Abscess"). In many cases, however, copious hemorrhage and an exsanguinated condition of the patient may contraindicate bronchoscopy, or require its postponement.

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BRONCHOSCOPY IN RELATION TO BRONCHOPULMONARY TUBERCULOSIS

Indications.—This subject is concerned with three classes of case: (1) Patients in whom a pulmonary symptom such as hemoptysis or cough has raised an unanswered question of tuberculosis. (2) Patients in whom the existence of pulmonary tuberculosis of the lower air-passages has been fully established by clinical features, physical signs, roentgenograms, and presence of tubercle bacilli in the sputum. (3) Patients with pulmonary tuberculosis on whom pneumothorax is proposed.

Patients having a pulmonary symptom, such as cough or hemoptysis, require diagnostic bronchoscopy as one of the important means of objective examination. This does not imply any other means should be neglected, all means are needed. Bronchoscopy is an addition to, not a substitute for, other diagnostic procedures. In patients known to have pulmonary tuberculosis, one or more diagnostic bronchoscopies should be done as part of the complete study of the case, just as complete study in any other region of the body accessible to specular examination is carried out. Whether or not treatment through the bronchoscopic speculum is indicated depends upon the presence and character of the lesions found at diagnostic bronchoscopy. The mere passage of the bronchoscope has never, in our experience, done any harm, therefore we may say in a general way that the presence of a tuberculous laryngeal or pulmonary lesion is not a contraindication. There are, however, a few contraindications. One is a hopeless case. When an expert in pulmonary tuberculosis regards a patient as beyond the possibility of recovery bronchoscopy may be considered as contraindicated, not because it would do harm but for the reason that there being no prospect of benefiting the patient the indications for bronchoscopy are lacking. In the curable stages, copious hemorrhage from pulmonary lesions known to be tuberculous is a temporary contraindication, unless bronchoscopic hemostasis is the objective. In case of small blood loss in patients who are known to have pulmonary tuberculosis but who are in fairly good general condition, bronchoscopy is not contraindicated. If the patient is not definitely known to be tuberculous, hemoptysis is not a contraindication except in case of

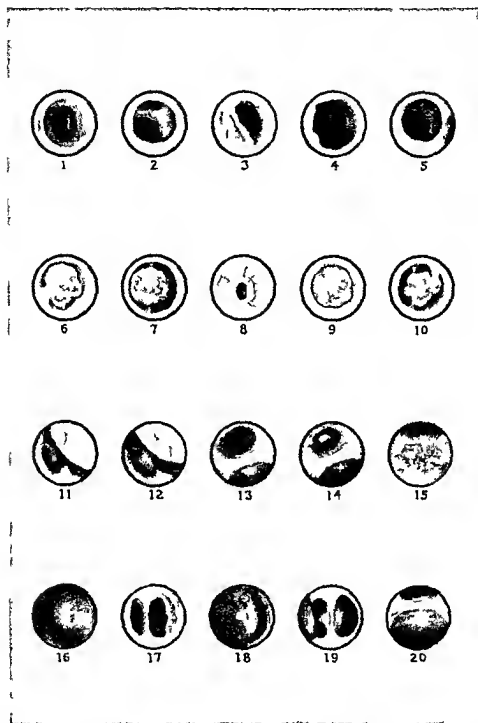


Fig. 472.—Bronchoscopic views in which pulmonary tuberculosis was present or required exclusion

1 Bronchoscopic view far down in the left lower lobe bronchus of a man aged forty-four with cardiac asthma that simulated pulmonary tuberculosis. The color of the mucosa is slightly cyanotic due to cardiac disease. Otherwise the mucosa is normal. The form is normal and the thin sharp spur between branch bronchial orifices is worthy of note. Bronchoscopically removed secretions were grossly normal and bacteriologically sterile. The symptoms, dyspnea and hemoptysis at one time deemed pulmonary and tuberculous were really due to the cardiac disease.

2 Chronic bronchitis and bronchiectasis of long standing as seen in the right bronchus of a man aged fifty-four. The thickening of the spurs between branch bronchial orifices is typical of chronic bronchial inflammation of many years duration (*cf.* 5). The mucosal vessels are not always so conspicuous as in this case. A practitioner had been in ecting the trachea with a silver nitrate solution and this ill advised treatment, though not the primary cause may have intensified the mucosal engorgement. Amelioration followed bronchoscopic aspiration combined

with medical care and management. Subsequent residence in Arizona rendered the patient symptom free so long as he remained there.

3 Cicatricial stenosis of the trachea in a woman aged forty one referred by a surgeon who had thirteen years previously, removed a cicatricial stenosis from the same region in this same patient by a well planned external submucous excision. For the recurrence here shown dilatation was undertaken bronchoscopically this gave perfectly normal breathing for ten years, when a second series of dilatations again gave excellent results. Five years later another series of dilatations was needed. The primary cause of the cicatricial stenosis was unknown, but exclusion of the possibility of tuberculosis was deemed advisable before undertaking treatment.

4 The same patient as shown at illustration 3. View three years after bronchoscopic dilatation. It is about the same today twenty years later. Three series of a few dilatations each have been given during this period.

5 Composite view down the normal right lower lobe bronchus of a woman aged nineteen suspected of having incipient pulmonary tuberculosis. The sharpness of the spurs between branch bronchial orifices is in marked contrast to the thickened spurs seen bronchoscopically in cases of disease especially diffuse, chronic mucosal inflammation (cf 2). There was a pin deeper down but at the level here shown the bronchial mucosa was normal.

6 Tuberculoma in a man aged forty-eight, who had been recently discharged from a sanatorium with a diagnosis revised from that of tuberculosis to that of allergic asthma. There was nothing in the history to suggest pulmonary tuberculosis earlier in life. The patient's chief complaints were wheezing, a cough (chiefly paroxysmal) and dyspneic attacks occurring chiefly at night and causing distressful gasping for air until secretions were raised. The physical signs were not definite. The fluoroscopist reported atelectasis of the left lung but films exposed on inspiration and expiration did not confirm the fluoroscopic findings. They showed a few calcified spots, otherwise no abnormality. Mirror laryngoscopy also revealed no abnormality. Bronchoscopy showed a pale nodular surfaced, pedunculated tumor attached to the left wall of the trachea at about 1 cm above the carina. At each inspiration the tumor was drawn downward rendering the pedicle visible (cf 7).

7 The same patient as shown in 6. Expiration caused the growth to flop upward and hide the pedicle from view. Bronchoscopic removal was not followed by recurrence up to the last examination four years later and there was no evidence of reactivation of the tuberculosis elsewhere in the lung. Histologic report by Dr Ernest W Willets: tuberculous granuloma.

8 Posttuberculous cicatricial stenosis of the trachea in a woman aged forty one sent to our clinic with a tentative diagnosis of allergic asthma. She gave a history of having been treated under a diagnosis of pulmonary tuberculosis when twenty one years of age. The symptoms on admission were wheezing, dyspnea on exertion and also nocturnal attacks of impending asphyxia with sudden awakening. These symptoms had been temporarily relieved by dietary restrictions based on allergic tests. Symptomatic recurrence led Dr Hobart Amory Hare to advise bronchoscopic examination. Harsh breath sounds and loud rales were heard all over the chest. The percussion note was impaired. Bronchoscopy revealed no abnormality other than the cicatricial stenosis. Specimens of secretions from below the stricture were repeatedly sterile. The condition of stricture was entirely cured by peroral bronchoscopic dilatation. As the lumen of the stricture was not extremely small it seemed probable that the lumen became occluded at times by secretions and exudates.

9 Tuberculoma in a man aged fifty two sent to the hospital with a diagnosis of allergic asthma. He was reported sensitive to a number of excitants and elimination of these had given real relief but awakening with severe nocturnal attacks of dyspnea persisted and for this the internist Dr James McKelvy had referred the patient to us for bronchoscopy. The chief complaints were dyspnea, wheezing, mucoid sputum sometimes blood streaked and paroxysmal cough. There were no physical signs except for a slight harshness of the breath sounds over both lungs. Mirror laryngoscopy revealed a larynx that would pass as normal. At bronchoscopy no evidence of laryngeal disease was found. A tumor mass was seen almost filling the tracheal lumen (cf 10).

10 Same patient as shown in illustration 9. At each inspiration the growth was drawn downward revealing a pedunculated attachment to the right tracheal wall. The growth was removed with bronchoscopic tissue forceps without much sense of resistance. Histologic report by Dr Ernest W Willets: epithelialized tuberculoma. There had been no recurrence and the patient was in good general health when last seen, two years later.

11 Lipoma of the trachea in a woman aged thirty six supposed to have pulmonary tuberculosis. The right bronchial orifice visible in the lower right segment was crowded backward and almost shut by the tumor. The left bronchial orifice was seen to be patulous but displaced. Its long axis was rotated about 20 degrees from the sagittal plane. The carina was crowded downward. The symptoms were dyspnea and unproductive cough. The only physical sign was tympany with absence of breath sounds on the right side. The left side was not far from normal. This sketch shows the position at the end of expiration (cf 12).

12 The same patient as in illustration 11. This sketch shows the appearance on inspiration. The growth is seen to be retracted by the inspiratory enlargement of the trachea. The carina was thinned by the inspiratory enlargement of the bronchial orifices. Slight movement of secretion indicated passage inward of some air at the maximum of the inspiratory phase. At each expiration a little bubble came out. This movement and the degree of flexibility of the tissues was in marked contrast to the rigidity seen in cases of malignant infiltration and was deemed suggestive of benign growth. After bronchoscopic biopsy showed the growth to be a lipoma (histologic report by Dr William Proescher) the tumor was removed bronchoscopically. The growth was sharply demarcated there was no involvement of the carina or bronchial orifice. They were simply distorted, crowded and displaced. A small recurrent growth was present at bronchoscopic examination four months afterward. Four years later there was no recurrence. The carina and bronchial orifices had returned to normal. It is noteworthy that we had here an example of check valve mechanism. By slight opening of the left bronchial orifice a little air was admitted

aneurysm The bronchoscopic following of a blood streak downward will usually lead to the source of the blood and to the correct diagnosis. In any case there is a possibility that a bronchoscopy may be done on a patient about to bleed dangerously, even fatally, under such circumstances the hemorrhage may be unjustly attributed to bronchoscopy instead of to coincidence. Pneumothorax, if spontaneous, is a temporary contraindication. Therapeutic pneumothorax, even bilateral, is not a contraindication.

Bronchoscopic Appearances of Tuberculosis—In the early stage, the mucosa of the trachea and larger bronchi may be of a dull velvety-red color. If there is much cough the mucosa may appear roughened. A few months later erosions may be visible and there may be patches of adherent secretion. These appearances are due to chronic tracheobronchitis and do not denote the presence of tuberculous lesions of the mucosa. Search of the tree may reveal bronchial orifices at different stages of development of tubercle. An early lesion will show a lumen diminished by a ringlike swelling of the mucosa and submucosa invaded by tubercles. The

breaking down of tubercles in this ring produces ulceration. The ulcer at first is rather flat but soon the typical chronic granulomatous process becomes exuberant and the lesion becomes elevated. This chronic granulomatous ulcer extends by continuity along the bronchial wall and because of its slow progress is the commonest of all bronchoscopic views. It is usually seen more or less edgewise. The visible elevated edge of the ulcer is pink. The lesion may heal leaving more or less fibrosis, according to duration of the granulomatous stage. It may be crescentic if only part of the periphery was ulcerated, or concentric if all the wall was involved (8, Fig. 472). The lumen of the stricture may be only 1 or 2 mm in diameter or there may be no lumen—a cicatricial bronchial atresia. In some cases after healing there remains an epithelialized granulomatous tumor sometimes pedunculated (6, 7, 9, 10, Fig. 472). This is uncommon. Usually the pinkish granuloma covering the ulcer with an advancing edge of submucosal tuberculous infiltration is seen occupying more or less of the bronchial lumen. Any remaining lumen may be seen in one of four conditions (1). It may remain open on

at each inspiration, prompt closure of the orifice at the beginning of the expiratory phase trapped air in the lung producing an obstructive emphysema of this lung.

13 Tuberculous abscess of the middle lobe in a woman aged eighteen. The middle-lobe orifice, seen in the upper segment, was rotated counterclockwise so that its long axis seemed about 20 degrees from the average normal horizontal position (cf. 15). The spur between the middle lobe and lower lobe orifices was similarly rotated and as here shown was greatly thickened, this was probably associated with peribronchial lymphatic involvement. The mucosa was swollen and red. The rather unusual intensity of redness was probably due to mixed infections. The velvety edema had encroached upon the lumen of the middle-lobe orifice.

14 Same patient as illustrated in 13. The pus had again welled up into the narrowed middle-lobe bronchial lumen. It continued thus to reappear until repeated aspirations with the aid of the tussive squeeze had emptied the abscess cavity. The last of the pus aspirated contained an abundance of tubercle bacilli though none had been found in the sputum.

15 Tuberculosis of walls of the lower lobe bronchus with complete filling of the lumen by tuberculous granulomas tinged with blood. The mucosa of the middle-lobe orifice shows beginning invasion. The spur is seen in the average normal horizontal position. The patient was recumbent, the middle lobe orifice is shown above the spur.

16 Gumma of the right bronchus. Under antisyphilitic treatment the gumma suppurated and cicatrized. The patient had been sent in with a tentative diagnosis of tuberculosis because of hemoptysis, slight fever, and loss of weight.

17 Same patient as in illustration 16. The cicatrices on the right segment mark the site of the gumma removed by internal medication.

18 Tracheal stenosis due to aneurysm. A reddish mass is seen bulging into the trachea from the left side, the upper border being about 3 cm. above the bifurcation. The surface of the mass is somewhat granular with a slight tendency to be nodular. No tracheal rings are visible in the mass. The pulsations of the mass were strong but apparently not expansile.

19 Same patient as in illustration 18. A 9 mm. bronchoscope compressed the reddish mass backward and gave a good view of the bifurcation. The carina was seen to be thin and flat. The right main bronchial orifice was apparently normal. The left bronchial orifice was crowded down somewhat by the overhanging proximal mass. The patient, under antisyphilitic treatment, lived for fourteen years before dying of asphyxia.

20 Early tuberculosis of the wall of the right lower lobe bronchus. The spur between the middle-lobe orifice (upper segment) and the wall of the lower lobe orifice was thickened. The mucosa of the lower lobe bronchus (lower segment) was dull and mottled, in contrast to the normal smooth mucosa of the middle lobe. Adherent thick pus had just been cleaned off with the aspirator before this sketch was made. Tubercle bacilli were found in this aspirated pus though they had not been found in the sputum.

inspiration and expiration (2) On inspiration it may open enough to admit air and immediately close at the beginning of expiration (Figs 473-474) (3) A small mass of exudate, pus, caseous material or debris may be seen to move upward on expiration and slap downward at the beginning of inspiration, both movements obviously the effect of the respiratory air current (4) The entire lumen may be occluded completely so that there is no opening on inspiration; there may or may not be bubbling out of a small quantity of pus from the occluded or the

of air. The slitlike lumen opens on inspiration enough to admit air downward, but at the very beginning of expiration the slit closes, imprisoning the air below. Obviously this causes emphysema in the subjacent lung. In all these valvular actions it is the pumping to and fro of the respiratory air column by the bellows like thoracic cavity plus the alternate expansion and diminution of the bronchial lumen that activates the valvular mechanism. The explanation of the occurrence of valvular action is not based on theory. The mechanisms were first discovered



Obstructive emphysema

Fig. 473



Giant cells

Fig. 474

Fig. 473—Obstructive emphysema of the right lung produced by tuberculous bronchial stenosis in a child aged twenty-six months, sent to the hospital with a diagnosis of suspected foreign body. Removal of tuberculous tissue caused disappearance of emphysema. The child progressed favorably under anti-tuberculous regimen.

Fig. 474—Photomicrograph showing tuberculous character of the tissue removed from the patient whose roentgenograms are reproduced in Figure 473.

strictured lumen. These are endoscopic evidences of valvular mechanisms causing emphysema or atelectasis (see under Bronchial Obstruction).

Another bronchoscopic view sometimes seen is a compression stenosis of the bronchus. The mucosa may or may not be normal, but the lumen is almost obliterated by the compression of a mass of tuberculous lymph nodes (Fig. 467). At a certain stage in the development of such a compression stenosis there is a check-valve action permitting only one-way passage

years ago in the author's clinic in connection with inspired foreign body^{1, 2, 3} and then were noted to occur often also in association with diseases of the lung and tracheobronchial tree. They will be fully understood from the diagrams illustrating the section entitled **Bronchial Obstruction**.

In case of *tuberculous abscess cavity* the same four valvular types apply to the fistulous orifice which is the inlet that affords communication between the abscess cavity and the particular bronchus. This orifice requires bronchoscopic

study to determine which of the four valvular mechanisms is in action a stop valve, a by pass valve, a one way valve admitting ingress of air but not egress (inflation), a one way valve permitting egress of air (and fluid) but not ingress (deflation), and drainage and collapse of the cavity)

Bronchoscopic Study in Relation to Therapy.—Here we are down to the fundamental of the bacteriology of tuberculous processes. In a particular case, do we want an abscess cavity ventilated and draining, that is with air going in and out at each respiratory cycle? Or do we want the cavity pumped empty of air and pus, and kept pumped out? Or is it desirable to have air pumped into a cavity so as to keep the cavity inflated. The answer to these questions depends largely on the predominant bacteria. Are they aerobic, that is, tubercle bacilli that die without air? Or pyogenic cocci that flourish whether air is present or not? From the previously mentioned observations it is seen that a cavity in a lung breathes, so to speak, in and out, if its orifice or fistulous channel is open to passage. It is in mechanical action, a relatively huge counterpart of an alveolus. In one respect it differs, namely, through the thin walls of a normal alveolus there is a free exchange of gases with the blood stream. If a group of alveoli, say a small segment of lung, is afflicted with a stop-valve obstruction the vascular walls will take up the gases. This is not true of an abscess cavity because its walls are thick and contain few vessels, moreover, what vessels may exist are surrounded by tissues and morbid products that retard or entirely prevent gaseous interchange with the blood stream. Consequently it may be stated that a normal lung, lobe, or segment may collapse from absorption of gaseous contents after stop valve obstruction, whereas a cavity may not be thus collapsed. If the mural barrier is thin or incomplete at any part, a greatly retarded absorption may result in a slow collapse. The bearing of the foregoing facts on collapse therapy is obvious. When artificial pneumothorax is contemplated it is well to determine the bacterial status. If pyogenic cocci predominate the advisability of collapse and imprisonment of these anaerobic organisms seems questionable. The only certain way of determining the bacterial status is by the laboratory examination of an uncontaminated bronchoscopically removed specimen of exu-

dative material from the focus of disease. If an abscess cavity is to be collapsed this determination is of utmost importance. To shut off ventilation and drainage from a cavity in which pyogenic cocci predominate may lead to disaster. The importance of the condition of the outlet of an abscess has been previously mentioned. An outlet for evacuation favors collapse of the cavity, but a one way valve effect may prevent collapse. Either condition can be corrected bronchoscopically and thus pave the way for collapse therapy.³

The great advances made in the treatment of certain phases of pulmonary tuberculosis by external surgery are not within the scope of this book. Suffice it to say that the surgeons doing this work constantly avail themselves of the important bronchoscopic information as to the endobronchial pathologic conditions in arriving at decisions as to whether or not to operate and in planning as well as in doing an operative procedure in operable cases.

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BRONCHOSCOPIC THERAPY IN TUBERCULOSIS

Tuberculosis of the bronchi appears to the bronchoscopist in the form of intensely red swollen mucous membrane, as ulcerations, as tuberculous tumors, or, in cases in which there has been healing as fibrous strictures. The ulcers vary in size and depth, some are merely

small patches and others include the whole circumference of the trachea or bronchus. It is difficult at times to estimate the longitudinal extent of the ulceration. The granulation tissue varies in amount from small elevated masses to tumors so large as to be obstructive. The strictures may be so extensive at times as almost or completely to close the main bronchus.

The method of treatment used will depend on the nature of the lesion found on bronchoscopy. The cases in which the tuberculosis manifests itself as a *general swelling of the mucous membrane* usually respond well to thorough shrinking by application of cocaine and adrenalin, removal of any imprisoned secretion beyond the narrowing by means of suction followed by the application of silver nitrate of various strength from 10 per cent up to 30 per cent.

The ulcerations are treated in various ways. Undoubtedly the simplest is the use of silver nitrate painted over the whole ulcerated area. The objection to the use of silver nitrate especially the stronger solutions, appears to be that the healing process following this treatment produces denser and firmer scar tissue. This may result in a very tight stricture of the bronchus and imprisonment of secretion beyond. To avoid this result the ulcers have been treated by the application of ultraviolet light by means of an electrode which can be introduced through the bronchoscope and applied for two or three minutes. This treatment has appeared at times to promote healing with a softer and more dilatable scar. The use of coagulation by diathermy on shallow tuberculous ulcers should be condemned as there is danger in these cases of too deep penetration and a subsequent secondary hemorrhage.

The treatment of *tuberculous tumors* depends somewhat on their size. Small elevated masses are easily removed by the use of strong silver nitrate solution. These can also be destroyed with safety by diathermy coagulation. Some have been removed with punch forceps. The objection to this is that any bleeding is likely to carry the tubercle bacilli to other portions of the lung and so spread disease. At times tumors will be found so large as actually to obstruct the mouth of a secondary bronchus or even a main bronchus. In the case of such obstruction their early removal is urgent because obstruction of a bronchus if prolonged always

leads to an abscess or bronchiectasis beyond the obstruction. It is in these cases that removal by punch forceps is, if ever, justifiable. The obstruction being relieved, the base of the tumor can then be cauterized with diathermy coagulation or silver nitrate.

The *scar tissue strictures* are a danger chiefly on account of the obstruction of the bronchus in which they occur. If such a stricture should occur in the trachea it may lead to death from slow strangulation. If the mass of scar tissue follows a tuberculous lesion which has included the whole thickness of the bronchial wall the control of the stricture will be very difficult indeed. On the other hand, if the fibrous stricture is due to the healing of an ulceration which has attacked only the mucous membrane the obstruction can be much more easily relieved.

These strictures have been treated by the use of glove stretcher dilators and by the passage of bougies. The objection to the use of these instruments is that they are likely to cause trauma and activate a quiescent lesion. It has been found that the use of a galvanic current through a copper electrode will soften the scar tissue and allow dilatation with comparatively little trauma. Proper electrodes can be made which can be passed through the bronchoscope and into a stricture, then a positive current of 3 ma. is used, at first for three minutes, the length of time of application being increased as the progress of the case seems to indicate.

In dealing with tuberculous diseases it will be found that *emergencies* will occasionally arise. One such would be the rupture of an abscessed lymph node into the bronchial tree. Such an accident seems especially liable to occur in children. These emergencies can be dealt with by at once passing a bronchoscope and aspirating the pus. If this cannot be done promptly death by drowning is threatened or a wide spreading of the infection may result.

Occasionally calcified nodes will erode their way into a main bronchus. There they will cause all the symptoms of an aspirated foreign body and it will be necessary to remove them with forceps in the same manner as other foreign bodies are removed.

Tumors and strictures always threaten obstruction of a bronchus and the imprisonment of secretion beyond the obstruction. Such an

occurrence manifests itself by fever, pain in the chest, and drying up of the hitherto abundant sputum. In such cases repeated bronchoscopies and aspiration of the secretion are indicated even though nothing immediate can be done to cure the main lesion. Occasionally a tumor in the mouth of a secondary bronchus will act as a ball valve and cause imprisonment of air in the tuberculous cavity beyond. When this happens even the most efficient pneumothorax or thoracoplasty will not close the cavity until the tumor is removed. Preceding the employment of pneumothorax or thoracoplasty for the closure of cavities a bronchoscopy is always indicated in order to make sure that there is no obstruction in the bronchus such as would prevent the lung from collapsing.

To sum up the treatment of tuberculosis of the tracheobronchial tree. In the first place immobilization of the lung should be secured if possible. Ulcerations should then be treated either by application of silver nitrate or the use of a mercury vapor lamp. If the ulcers in healing cause stricture of the bronchus, the fistula must be kept open until the lung beyond has been dried up by repeated aspirations. The strictured bronchus can be kept open by the application of a positive galvanic current using copper electrodes. At times a stricture will completely close a bronchus. There is no danger in this if the lung beyond is dry. If the lung cannot be dried up by repeated aspirations, threatened closure of a stricture must by all means be resisted, using preferably galvanism through a copper electrode. The treatment of tuberculous tumors, especially if they are obstructive, consists in their destruction by coagulation. It is thought that this procedure will be less likely to result in the spread of the disease than will their removal by forceps. As to the frequency of treatment of tuberculous lesions through the bronchoscope, in most cases every two weeks will be found sufficient. When there is great tendency to greater secretion beyond the narrowing bronchus weekly treatment may be necessary.

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BRONCHOSCOPY IN RELATION TO NONTUBERCULOUS PULMONARY ABSCESS

A nontuberculous pulmonary abscess may be defined as a localized collection of pus in a cavity formed by disintegration of the tissues. The term has been so loosely used that the literature is chaotic, rendering analysis of statistics laborious and inaccurate. When, owing to bronchial obstruction, pus collects in natural passages the walls of which have not broken down the condition is not abscess, it might be called "drowned lung."¹ The term "abscess" should not be applied to pus collections in cases of bronchiectasis or suppurative bronchitis, nor should it be used synonymously with the loose term "pulmonary suppuration."² Attention to these distinctions would enormously increase the value of statistics.

Indications for Bronchoscopy.—The bronchoscope is a bronchial speculum. It bears the same relation to intrathoracic disease that the vaginal speculum bears to intrapelvic disease, namely, that its use as one of the methods of examination is called for in every case. The results of all the various methods of examination will determine whether or not the bronchial speculum will be needed in carrying out the treatment decided upon as indicated in the particular case. For clearness the various indications may be considered under five stages of pulmonary abscess, namely, (1) prophylactic, (2) diagnostic, (3) acute, (4) subacute and chronic, (5) preoperative and postoperative.²

The prophylaxis of pulmonary abscess due to bronchial obstruction (*q. v.*) is one field in which treatment through the bronchial speculum occupies a unique position of greatest usefulness, as obviously demonstrated in cases of obstruction due to foreign body. Most of such cases are potentially productive of pulmonary abscess, but early removal by bronchoscopy is successful in 98 per cent of the cases both as to removal and as to prevention of suppuration.³ The group of cases of obstruction due to endogenous foreign body (*i. e.*, pathologic products) is not so large but is large enough to place bronchoscopy as first among the means of prophylaxis in nearly all cases. The restoration of ventilation and drainage by bronchoscopic removal of obstruction reestablishes the defensive power of the lungs. The resistance to infection by the endobronchial route is natu-

rally high ¹ Of course, in such cases, it must always be considered as associated with medical care and management ⁴ In *diagnosis* the bronchoscope has the important duty of supplying the essential clinical facts of the living pathologic conditions present in the lung of the particular patient under diagnostic study. Objective appearances as well as uncontaminated specimens of tissues and exudates are definite clinical data of utmost importance, for example, cancer and tuberculosis require consideration in the differential diagnosis of every case of pulmonary abscess. Bronchoscopic biopsy supplies absolute certainty in such cases. The predominant fungi, cocci, bacilli, or other micro-organisms must be determined in every case of pulmonary abscess, the uncontaminated specimens obtainable with ease, certainty, and precision by bronchoscopy are invaluable in these determinations. They supply the only reliable basis for decision as to best treatment by chemotherapy, as well as by surgery or other means. In *acute pulmonary abscess* bronchoscopy may initiate or provide adequate drainage, permitting the abscess to go on to spontaneous closure and healing. It is in this stage, however, that the patient must be watched carefully, so that if improvement should cease, other measures may be resorted to without delay and serious complications such as gangrene, extensive progressive suppurative pneumonitis, and bronchiectasis, thus be prevented. This period extends roughly to about four or six weeks ² In *subacute and chronic abscess* the patient should be put upon what we call the *conservative bronchoscopic regimen* for a trial period. If definite progress toward healing of the abscess is not soon noted, other methods of attack should be adopted. For example, in a series of eighty-seven patients with pulmonary abscess under this regimen, which includes bronchoscopy, thirty-three patients were cured and seventeen were improved, but it was found inadvisable to continue the conservative regimen longer than four or six weeks, and not even this long, if favorable signs were not in evidence soon after beginning of the system of conservative measures ² The conservative bronchoscopic regimen comprises a number of different but closely related procedures, any or all of which may be employed in the treatment of any individual patient. These practices are: (1) routine supportive care including bed rest, preferably under out door conditions, nursing,

balanced diet, vitamin supervision, and symptomatic treatment, (2) postural drainage, (3) bronchoscopic procedures, (4) transfusions, (5) vaccines, penicillin intravenously, neoarsphenamine, sulfonamides, and other special medications in particular cases. The role of bronchoscopy in this regimen is the restoration of ventilation and drainage by the following means: (a) The load is taken off the cilia by frequent aspiration of pus—weekly, biweekly, or oftener if necessary. This prevents stagnation and the “septic tank processes” ⁵ that lead to bronchiectasis (qv) or unhealed abscess. (b) Removal, by swabs or forceps, of semisolid exudates, sequestra, or other endogenous foreign bodies. (c) Removal of obstructive granulations and granulomas with cupped forceps. (d) Gentle dilatation, rarely necessary, to enlarge the lumen of the orifice of the abscess. Important as is the just outlined bronchoscopic restoration of ventilation and drainage, it would be a great mistake to depend upon it alone, or, in ambulant cases, to allow the patient to neglect other factors in the regimen, as he is prone to do. It is of utmost importance to restrict his activities by stipulated hours of rest in bed, to build up a reserve of resistance. If this be not done he will use up any improvement in increased activity. Of course, in bedfast cases, all factors of the regimen are under control. In the clinic whose statistics are quoted earlier in this paragraph, there are consultations among the medical, surgical, roentgenologic and bronchologic departments. For convenience in obtaining these consultations, all cases are presented and discussed at the weekly “Chest Conference” ²

Synergistic Bronchoscopic Aspiration of Pulmonary Abscess—In addition to the technique of bronchoscopy previously given certain points require emphasis. The *tussive squeeze* ⁶ is of utmost importance during the bronchoscopic aspiration. The bronchoscope does not, and need not, reach the abscess cavity. It is not necessary, nor even desirable, that the aspirating tube should reach it. It cannot be too strongly stated that, as demonstrated over twenty years ago, ⁶ bronchoscopic aspiration removes the pus as fast as it is forced upward into the larger bronchi by the tussive squeeze. Therefore, premedication and local anesthesia if used at all must be minimized so as not to lessen the cough reflex ^{6, 7} In order to emphasize the necessity for the cooperation of the

tussive squeeze we have called the procedure synergistic aspiration

POSTTONSILLECTOMIC PULMONARY ABSCESS

Posttonsillectomic pulmonary abscess is a distinct type of abscess and also a distinct type of postoperative pulmonary complication. It was at first supposed to be due to inspiration of blood and infective secretions from the mouth and especially infective material squeezed out of the tonsils in the course of their removal. This was disproven by bronchoscopic observa-

tion enough between the operation and the abscess formation for the lymphatic route to be suggested.⁸ The symptoms are usually of sudden onset and in most cases begin within a few days or a week after operation; a few occur after various periods up to about four weeks; six weeks are apparently the extreme limit. A chill, fever, cough, foul expectoration, pain in the chest, hemoptysis, anemia and leukocytosis are the usual symptoms.⁸ At bronchoscopy a bronchus is usually found filled with foul pus. When this is aspirated the pus is seen to come from one branch, bronchial orifice, or from a ragged



Fig. 475.—Roentgenogram (left) showing a slight shadow of atelectasis and drowned lung due to the seven months' sojourn of a tonsil in the left bronchus. Notwithstanding the probable presence of blood clots and the prolonged obstruction of a bronchus by the tightly fitting plug of tonsillar tissue filled with septic material, no ill health sufficient to keep the child out of school resulted, and not one of the clinical features of posttonsillectomic pulmonary abscess was present. The symptomless interval of two months, the mildness of the symptoms when they did develop, and the prompt recovery after bronchoscopic removal were all features commonly seen in nonseptic bronchally lodged foreign bodies. They are in marked contrast to the fulminating onset of posttonsillectomic pulmonary abscess, and they point to the previously noted great defensive power of the lung against invasion by way of the bronchial mucosa. Roentgenogram (right) of the same patient showing the complete disappearance of the pathologic shadow in three weeks after bronchoscopic removal of the tonsil that had been in the lung for seven months. The inset is from a photograph of the tonsil reduced in the same proportion as the roentgenogram.⁸

tions which demonstrated the existence of a powerful defense mechanism against infection of the lung through the bronchial wall.¹ For example, in hundreds of cases septic material like nasal and alveolar bones, meat, teeth, parts of instruments used on foul root canals and septic tonsils, even portions of the tonsil itself, did not cause pulmonary abscess.⁸ A tonsil remaining as long as seven months in a bronchus did not cause an abscess (Fig. 475). The pathologic process is due to a septic infarct lodging on the other side of the bronchial barrier. The route is probably the blood vessels in most cases. In only a small proportion of cases is there time

for an orifice in a bronchial wall. The diagnosis is made by roentgen ray examination and bronchoscopy. These examinations are urgently called for by the development of any of the symptoms just mentioned.^{2, 8} Treatment is by synergistic bronchoscopic aspiration and bronchoscopic maintenance of ventilation and drainage, along with the entire regimen of conservative treatment given in connection with nontuberculous abscess. Opium derivatives, all other sedatives, and atropine block drainage and must be absolutely forbidden.¹⁰ External operation may be required as in nontuberculous abscess, but the records show it is

less often required in these postionsillectomic cases Possibly this is because the tonsillec-
tomist promptly resorts to bronchoscopic pro-
motion of drainage Probably for the same
reason the *prognosis* is better than in any other
form of pulmonary abscess except in that due
to inspired foreign body ¹

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POSTOPERATIVE PULMONARY ATELECTASIS WITHOUT PNEUMOTHORAX

Postoperative pulmonary atelectasis without
pneumothorax is the name given to pulmonary
atelectasis occurring after an operation on any
region other than the thoracic It occurs most
frequently after abdominal operations, and
bears little or no relation to inhaled anesthetics

as shown by not infrequent incidence after op-
erations under local and spinal anesthesia Lee¹
demonstrated that the condition was due to
obstruction by thick secretions accumulated
during operations especially prolonged op-
erations As in other forms of foreign body ob-
struction (*q v*) the pulmonary atelectasis occurs
without pneumothorax and may be favored by
the operative admission of air to the abdomen
The stagnation of secretions is favored by the
shallow respiration and the suppression of
cough, deep breathing and cough cause pain in
the abdomen, so that the patient naturally
avoids them The chief symptoms are the sud-
den onset usually within forty-eight hours after
operation, elevation of temperature, pulse and
respiratory rate, shallow respiration dyspnea
cyanosis, suppressed cough pain in the chest or
abdomen or in both The diagnosis is by these
symptoms accompanied by physical signs of
atelectasis (*q v*) The best prophylaxis is post-
operative aspiration of secretions by the anes-
thetist and absolute avoidance of premedication
with opiates and atropine ² Frequent change of
the patient's posture, encouragement of deep
breathing and cough are helpful prophyl-
actically

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BRONCHIECTASIS

Disease of the bronchial wall associated with
dilatation is known as bronchiectasis It is a
common disease

Etiology—Bronchial obstruction and infec-
tion are the chief primary causes Viscosity of
bronchial inflammatory exudates is a primary
factor discovered by bronchoscopy Once es-
tablished the pathologic conditions known as
the bronchiectatic septic tank are the chief per-
petuating or secondary factors ¹ Viscid ad

herent secretions of acute or chronic bronchitis supply the initial obstruction in most cases. The modern bronchoscopic treatment of chronic, subacute, and residual bronchitis (*q v*) by seriatim examination of the orifice of each segmental bronchus has abundantly demonstrated that obstruction of one small orifice is the rule rather than the exception, and obviously we have here an overlooked etiologic factor in bronchiectasis. Foreign body is occasionally an etiologic factor, but even in such cases the viscid inflammatory exudates greatly increase the degree of obstruction and are powerful factors.

Pathology.—Bronchoscopic studies revealed a group of pathologic processes and conditions to which has been given the name of the *bronchiectatic septic tank*.¹ This term is meant to designate not only a receptacle for septic materials but a container where bacterial processes change the character of the contents in a way comparable to the processes in what the sanitary engineers call a "septic tank." Bronchoscopic studies show that these changes in the secretion constitute nature's way of rendering expulsion of the secretion easier by reducing the viscosity. Unfortunately, however, the changes also render the material more irritating and the stagnation of this irritating material promotes pathologic changes in the bronchial walls, and thus constitutes an important etiologic factor in bronchiectasis. The bronchiectatic septic tank constitutes a fearful price to pay for drainage. Sputum in bronchiectasis comes up without revealing its original viscosity and it separates in layers in a glass receptacle, just as does the material in a septic tank. But the bronchoscope revealed the fact that the primary exudates in all inflammatory conditions of the bronchial mucosa are of extremely high viscosity and are so tenaciously adherent that they are extremely difficult to expel by the normal peroral means of pulmonary drainage, namely, ciliary action, tussive squeeze, and belch blast.² Autopsy shows the tissue changes in the bronchial walls to be a destruction of the fibro-elastic tissue associated with perichondritis and chondral necrosis. There are cylindric, saccular, or fusiform dilatations—fibrotic cavities partly filled with foul pus. Fibrotic changes in the mucosa are associated with destruction of glandular elements, in patches. There may be areas of pneumonitis, atelectasis, or emphysema.

Symptoms.—Cough, not constant, but at intervals, is productive of foul sputum, usually in rather large quantities. It is sometimes blood-tinged or may contain brownish clots. Hemoptysis occurs in some cases. The breath is foul. Dyspnea on exertion is noted when the affected area is large or the patient is inactive by habit. Clubbing of the distal phalanges with "watch-crystal" nails, the so-called "hippocratic fingers," are characteristic in cases of long duration. The earliest symptoms are seemingly so insignificant that they are not taken seriously by either patient or physician, usually nothing more than a slight occasional cough, productive of a little sputum not noticeably malodorous. After some months a slight odor is detectable in the morning sputum. These early symptoms are usually the sequela of an acute infection, often a so-called "common cold."

Bronchoscopic Appearances.—On first insertion of the bronchoscope through the glottis of a patient with fully-developed bronchiectasis there comes out through the bronchoscope a gush of foul pus of a peculiar unmistakable odor. The bronchoscopic aspirator will evacuate first a pus of creamy consistency. After this superjacent pus has been thus removed, the aspirated pus is thicker, the last or residual pus is of high viscosity and adhesiveness. Making due allowance for the obtunding of the olfactory sense, the odor of the breath coming through the bronchoscope is notably less. Having cleared the field by aspiration a variety of images may be noted. There are dilatations, stenoses, cicatricial areas, erosions, granulations, granulomas, with some intervening patches of chronic inflammatory mucosa. Branching vessels may be visible. The walls—instead of the normal even, fair curves, ovals, and circles—are irregular in form and may be ragged (3, Fig 476). Branch bronchial orifices may be gaping, occluded, strictured, or discharging.

Diagnosis.—In a fully-developed case of bronchiectasis the bronchoscopic appearances and odor of breath through the tube are diagnostic. In the early cases, however, bronchography is essential and it should never be omitted from the diagnostic study of the case.³

Prophylaxis.—It is safe to say that the incidence of bronchiectasis could be reduced to relative rarity if every patient with tardy recovery from acute infections of the lower respiratory tract were promptly given the benefit of

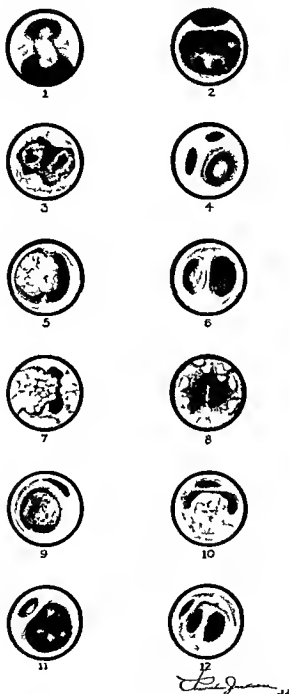


Fig 476 —Photoprocess reproductions of oil-color drawings illustrative of endobronchial disease as seen through the bronchoscope in the living patient

1 Pus in the swollen inflammatory orifice of the middle-lobe bronchus no evidence of air passing in or out as seen six weeks after tonsillectomy Cure by bronchoscopic aspiration This case is a demonstration of *bronchoscopic prophylaxis* of chronic abscess by arrest of the suppuration in the acute stage

2 Abscess of the right lower lobe in a boy aged nineteen years The middle-lobe orifice anteriorly (upper part of field, as seen in recumbent patient) is nearly normal The mucosa of the lower lobe bronchus is intensely inflammatory and covered with bloody granulations among the granulations pus is stagnant Perfect restoration of ventilation cessation of suppuration, and a normal state of the mucosa followed 15 bronchoscopic aspirations.

3 Appearances in a bronchiectatic septic tank on first examination with the field cleared of stagnant pus

bronchoscopic aspiration. So-called unresolved pneumonia, residual interstitial pneumonitis and other pathologic conditions may be placed in the same category. Bronchoscopy in addition to its prophylactic value often demonstrates that the pathologic conditions present were not pneumonic in origin. Unquestionably many forms of pneumonia, virus pneumonia for example, are primarily diseases of the bronchial system with atelectasis, emphysema, interstitial pneumonitis, and other secondary changes in the lungs. Infective disease of the nasal cavities and nasal accessory sinuses must not be neglected but this is not enough. Prompt bronchoscopic arrest of the lingering remainders of acute infections of the lower respiratory tract, localized as obstructive of one or more branch bronchial orifices, is absolutely essential. Those thick tenacious secretions occurring as localized obstructions with which peroral pulmonary drainage is unable to cope, must be removed by synergistic bronchoscopic aspiration. Along with this must go a strict conservative regimen to maintain

maximum resistance. This regimen should include (1) physical rest, (2) good ventilation, (3) sunshine, (4) regulation of diet, (5) maximum vitamin intake constantly maintained, (6) any chemotherapy or other medication that may be indicated, prohibition of all sedatives and antituberculous. Unless the regimen is strictly enforced the patient will use up increased energy in increased activity instead of utilizing it to build up a reserve of resistance. If this plan were followed after every acute infection of the tracheobronchial tree that shows a tendency to linger, bronchiectasis would become a rare disease. We have abundant clinical records affording documentary evidence of this clinical fact.

Treatment and Prognosis—Bronchiectasis in an early stage can be cured in many cases by the conservative regimen described in connection with prophylaxis. In fully developed bronchiectasis limited to one lobe, lobectomy has given satisfactory results. In cases of bilateral involvement, major surgery is justifiable if the patient chooses to have it done. Palliative treatment can make the patient comfortable and obviate

by bronchoscopic aspiration. The ragged irregular orifices of two fistulous bronchi are seen at the bottom of a bronchiectatic dilatation. The mucosa is cicatricial in some parts, eroded in others. After 12 bronchoscopic aspirations the patient, a man aged twenty-four years, gained weight and the foul odor was ameliorated to an extent that enabled the patient to recover his morale and return to work, though he is not cured. He comes in occasionally for bronchoscopic aspiration.

4. Inflammatory stenosis, subacute, at the orifice of a posterior external branch of the lower lobe bronchus in a girl aged seventeen years, with cough, fever and weight loss. There was no ingress or egress of air with respiration. After the fourth bronchoscopic aspiration bubbles of air were seen to enter and emerge from the bronchial orifice. After 18 bronchoscopies the pathologic conditions and the symptoms had completely disappeared.

5. Bronchoscopic appearances of a lipoma obstructing the left main bronchus of a woman aged sixty-four years. Productive cough and hemoptysis ceased soon after bronchoscopic removal of the tumor. The result in this case was due solely to endoscopic treatment based on bronchoscopic diagnosis as to the lesion present and it would seem that the word *cure* might be properly applied.

6. Productive cough, hemoptysis and wheezing ceased after bronchoscopic removal of the obstructive granulation tissue shown in the right bronchus, followed by a series of bronchoscopic aspirations. A good illustration of the restoration of the defensive powers of the lung after endoscopic treatment based upon bronchoscopic study of the pathologic conditions present in the particular case.

7. Recurring multiple papillomas in the trachea that produced nocturnal attacks of dyspnea and wheezing suggestive of asthmatic bronchitis. A number of bronchoscopic removals have given complete relief for a time but recurrences have not ceased. There were no papillomas in the larynx. The amount of the purulent sputum increases as the growths repopulate between removals.

8. Multiple osteomas of the trachea (*tracheopathia osteoplastica*) associated with pulmonary suppuration and malignant disease of the pleura and left lung.

9. Adenoma obstructing the lower lobe bronchus and producing atelectasis in a girl aged fifteen years. All the symptoms, productive cough, fever and loss of weight, disappeared after bronchoscopic removal of the obstructive tumor. They reappeared with a recurrence of the tumor and again disappeared after bronchoscopic removal of the recurrence.

10. A sessile tumor mass obstructing the right lower lobe bronchus and producing an area of suppuration and obstructive atelectasis in a woman aged twenty-five years. The symptoms, productive cough, weight loss and fever, disappeared after bronchoscopic removal of the obstructive tumor.

11. Lobulated tumor obstructing the left lower lobe bronchus of a woman aged thirty-two years. Suppuration, cough and fever disappeared after bronchoscopic removal of the growth. The tumor histologically was an adenoma. No recurrence of the neoplasm or the suppuration had taken place at the end of six months (see 12).

12. The same patient as illustrated at 11, appearances six months later. Slight irregularity of the mucosa and a somewhat cicatricial appearance were seen at the site of removal of the tumor (upper right hand quadrant).

his being a social outcast. The odor of his breath can be rendered unnoticeable at a meter's distance, his expectoration can be limited to certain periods, and his life expectancy can be prolonged, usually, to actuarial averages.

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EMPHYEMA

The pleural cavity cannot be efficiently drained by peroral bronchoscopy, but the records of our clinic show that obstructive bronchial lesions are often important etiologic and perpetuating factors in emphyema. In such cases the patient will never get well so long as upward bronchial drainage is blocked. Our records also show so many cases in which bronchoscopic examination has contributed so largely to diagnosis, and occasionally so much to the treatment, that we feel justified in advising diagnostic bronchoscopy in all cases of chronic emphyema. Obstruction to peroral bronchial drainage, foreign body, broncholith, and benign growth are some of the conditions revealed and efficiently treated by peroral bronchoscopy. In not a few cases of emphyema the diagnosis of malignant tumor has been unexpectedly made by peroral bronchoscopic biopsy.¹

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BRONCHOSPASM

Bronchospasm is the name given to spasm of the bronchial musculature. It is also called *bronchospasm*, *bronchotetany*, *spasmodic bronchostenosis*. Its incidence as a pathologic condition cannot be determined. It seems to be common though probably not so common as one would infer from the literature, inasmuch as spasm of invisible viscera is usually on the fallible basis of inference. In many such cases we have found thick tenacious obstructive secretions the bronchoscopic removal of which cured the symptoms. One frequent factor in causation of bronchospasm is anaphylactic in character and is considered under "Allergy of the Nose and Paranasal Sinuses" and under "Diagnosis and Treatment of Hay Fever." A reflex nervous etiologic factor is believed to cause asthma as well as allergic conditions. As to the pathology, little is known. There is no morbid anatomy. The trachea has involuntary muscular fibers running vertically in the posterior membranous portion, and well-developed fibers completing the trachealis muscle connect the free ends of horseshoe shaped rings. The action of this muscular layer is often bronchoscopically seen to contract causing a mounding of this wall into the lumen of the trachea (Fig 363). In the bronchi the muscular layer is a complete ring, and its action is seen during cough as a concentric diminution of lumen. In asthma and other conditions believed to be associated with bronchospasm it is difficult to distinguish between this concentric diminution and a spasmodic contraction of the bronchial muscular layer except as a continuous abnormally lessened diameter of the bronchial lumen on full expiration.

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TUMORS OF THE TRACHEA AND BRONCHI

General Considerations.—Both benign and malignant tumors are encountered in the tracheobronchial tree. A benign tumor is one that does not metastasize and whose cells do not infiltrate among adjacent normal cells. "Benign" is not synonymous with "innocent," inasmuch as such a growth may cause death primarily by asphyxia, or secondarily by the suppurative disease caused by its obstruction to ventilation and drainage. A malignant growth is one that invades and infiltrates adjacent tissues locally, and metastasizes.

Prior to the advent of bronchoscopy, primary growths of the tracheobronchial tree were regarded as exceedingly rare. This was owing to the fact that at autopsy the small primary growth was not found, it was obliterated by the extensive suppurative disease secondary to bronchial obstruction. Nor were these small tumors encountered by the surgeon in the pre-bronchoscopic days, though surgical treatment was often utilized in secondary suppurative conditions. The medical diagnosis in such cases was pulmonary abscess or bronchiectasis, and this was in some cases correct as applied to the secondary pathologic changes. Bronchoscopy has shown that histologically nonmalignant tumors are relatively common, though not quite so common as malignant ones.

Etiology.—Tumors of the tracheobronchial tree are similar in origin to the same tumors occurring elsewhere. The cause of granulomas is, in some cases, specific infection, for example, tuberculosis (*q v*), syphilis, spirochetosis, blastomycosis, and actinomycosis. In two instances in the author's clinic granulomatous masses were removed from the orifice of the right bronchus of men who had been gassed three and six years, respectively, prior to bronchoscopic discovery of the tumor. In four cases of papilloma of the bronchi there was nothing to indicate an irritant factor. Air cysts are congenital, and, of course, teratomatous cysts are also, though other factors may be concerned in the associated secondary changes. The irritation of tobacco smoke, as well as other forms of fumes and smoke, has been mentioned as an etiologic factor in malignant tumors of the bronchi, but this has not been conclusively proven. In several of our cases, carcinoma has developed in the same lobe of the lung in which

a foreign body was lodged for a prolonged period (Fig 477).

Pathology.—Our records include histories of the following tumors and tumor-like conditions: angioma, hematoma, adenoma, myoma, myxoma, papilloma, fibroma, fibrolipoma, lymphangioma, lymphadenoma, lymphoblastoma, lipoma, echondroma, osteoma, chondrosarcoma, neurofibroma, retention cyst, amyloid tumor, aberrant thyroid tumor, specific granuloma, nonspecific granuloma, carcinoma, sarcoma, Hodgkin's disease. Chondromas and osteochondromas are benign, but may develop malignancy, and undergo sarcomatous or other changes. Edematous polyps and other more or



Fig 477.—Carcinoma developed in the left upper lobe bronchus twenty two years after a piece of shrapnel had penetrated this lobe. Diagnosis was established by bronchoscopic biopsy. Pneumonectomy was successfully done by W. E. Burnett. The patient is still well five years after operation.

less tumor-like inflammatory growths which are occasionally encountered are of the utmost importance because of the atelectasis, drowned lung, and suppurative conditions they produce. Histologically these growths do not differ greatly from the same growths encountered elsewhere, but adenoma of the bronchus (*q v*) has some peculiar characteristics. One pathologically important peculiarity of all growths in the tracheobronchial tree is that, though small, they set up diffuse destructive secondary changes in the distal portion of the lung, out of all proportion to their size. When ventilation and drainage are stopped by a tumor occluding a bronchus the few bacteria present in the distal area become active, suppurative inflam-

mation develops and becomes progressively worse. The suppurative stage often comes on quite suddenly with an attack of influenza or other acute respiratory infection. The obstruction of a bronchus by a tumor is of three types mechanically similar to the valves in pumps namely a stop valve a check valve or a by pass valve. It should be stated as a pathologic fact that the obstructive type of atelectasis produced by stop valve obstruction is different from atelectasis due to pneumothorax in that suppuration soon develops in the atelectatic area if the obstruction is not removed. It should also be mentioned that a peribronchial tumor can cause a check valve mechanism of the expansile type producing an obstructive

tumors of the trachea. Hemoptysis is a common symptom of bronchial tumor and is quite constantly present early in adenoma and carcinoma.

Diagnosis—Complete and accurate diagnosis requires orderly procedure (1) anamnesis with careful record of symptoms (2) general medical examination with record of all physical signs (3) blood count and serologic test (4) repeated sputum examinations (5) roentgen ray examination (6) diagnostic bronchoscopy.

Physical signs in the case of uncomplicated nonobstructive bronchial tumors are normal. Just as soon as a growth reaches a size sufficient to cause the slightest degree of obstruction however the changes in physical signs are definite

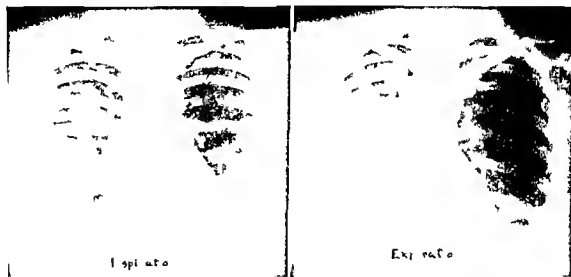


Fig. 478—Obstructive emphysema of the left lung due to check valve obstruction by a bronchial carcinoma. Note that the emphysema is most apparent on expiration because the unobstructed lung empties completely; the left lung remains inflated. The heart shifts over to the unobstructed side.

emphysema as well as later a stop valve mechanism producing an atelectasis just as an intrabronchial tumor can.

Symptoms—The symptoms depend upon the stage at which the patient is seen. In the beginning there are no symptoms. A wheeze heard at the open mouth, not at the chest wall, is usually the earliest symptom, and this wheeze has often led the physician to make an erroneous diagnosis of asthma. Cough and slight mucoid expectoration are noted as the tumor grows, usually becoming more troublesome when the growth occludes the bronchus. Dyspnea independent of exertion does not occur from obstructive atelectasis of a lobe or even of an entire lung, but it does occur relatively early in

and of great importance. The earliest of the signs of bronchial obstruction, whether caused by a tumor or a foreign body, are the asthmatic wheeze heard at the open mouth and the physical signs of by-pass valve obstruction, chiefly harshness of breath sounds distal to the tumor. Then come the physical signs of check valve obstruction producing emphysema (or in rare instances atelectasis). These also are distal to the tumor. It is not necessary to describe these signs here, since they are fully described in connection with foreign body, but it is necessary to say, and to say it emphatically, that when the physical signs show atelectasis or emphysema, bronchial tumor should be placed first or along with foreign body among the diag-

nostic possibilities. Diagnostic bronchoscopy is the only means for determining the character of the lesion or substance causing the obstruction. Of diagnostic importance equal to that of the physical signs and the diagnostic bronchoscopy is the *roentgen-ray examination*. It is true that small endobronchial benign tumors do not cast a shadow, but fluoroscopy demonstrates the differentials in aeration present in all cases of check-valve or stop-valve obstruction. Moreover, in the case of larger growths, the growth usually does show as a shadow in the roentgenogram.

Certainly biopsy material, when obtainable, should be carefully studied, but we must not concentrate our attention so closely on biopsy as to neglect the other aspects of the bronchoscopic and clinical picture. If the accessible portions of the tracheobronchial tree are found to be entirely normal, so much the better, the surgeon knows that in such a case he can amputate any of the larger bronchi and not leave tumor tissue behind, though he must proceed without knowledge of the histopathologic character of the lesion, unless biopsy material has been obtained by needle aspiration.



Fig. 479.—Obstructive atelectasis of the right lower lobe due to bronchial adenoma in a female nineteen years of age. Pneumonectomy was done in this case because of extensive suppurative changes in the distal portion of the lung. This patient is still quite well four years after the operation.

Bronchoscopic examination will generally demonstrate the presence of a benign tumor of the tracheobronchial tree, but in only about 75 per cent of the cases of carcinoma will the lesion be visualized. Nevertheless, the bronchoscopic findings, whether or not a tumor is demonstrated, are *always* important. Bronchoscopy should inform the surgeon as to the presence or absence, and, if present, the location and extent, of a neoplastic process in the accessible bronchi, the mobility or fixity of the tracheobronchial tree, and the histologic character of the lesion, when biopsy is possible. Perhaps a little too much emphasis has been placed upon biopsy as the reason for bronchoscopy in the

past. Of course, when the first bronchoscopy reveals no endobronchial tissue for biopsy, or tissue removed for biopsy proves to be normal histologically, bronchoscopy should be repeated after a suitable interval, because very often either intrusion or extension upward from the small branch bronchus in which the tumor originates will have taken place, and biopsy will be possible. Exploratory thoracotomy should be done, and lobectomy or pneumonectomy considered, however, without waiting for biopsy confirmation, if a fairly certain clinical diagnosis of malignant tumor can be made.

Treatment—Endotracheal and endobronchial benign growths of any size can be removed

endoscopically. Vascularity unless there has been exsanguinating hemorrhage is no contraindication. Carcinoma and sarcoma of the

the exception of lymphoid tumors call first for evaluation of operability. If lobectomy or pneumonectomy cannot be done, palliative roent



Fig 480 A tumor of the trachea (lymphoblastoma) is clearly shown in the lateral roentgenogram. Right the appearance after its removal by tracheoscopy using forceps and surgical diathermy. (See Fig 481.)

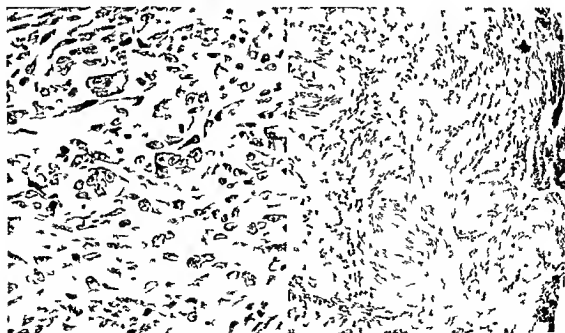


Fig 481 —Photomicrographs showing lymphoblastoma of trachea. (See Fig 480.)

trachea must be treated by deep roentgenotherapy and interstitial irradiation by means of radon seeds introduced through the tracheoscope. Malignant tumors of the bronchi with

genotherapy can be considered but will probably offer little benefit except in the lymphoid tumors.

Large peribronchial compressive benign

growths are dealt with by external surgery. This applies particularly to neurofibromas and to dermoid cysts causing suppurative destruction of pulmonary tissue. In cases of air cyst, treatment is not indicated except when the interior is infected and suppurative. Roentgen ray treatment externally applied is usually effective only in lymphoma, lymphosarcoma, or Hodgkin's disease.

Prognosis—Some benign tumors of the trachea and bronchi are fatal, primarily by asphyxia or secondarily by the sequential suppurative disease of the lung, but almost all can be removed bronchoscopically if diagnosed early, and the prognosis as to life is good. The prognosis of benign adenoma is good as to life, even in the few cases in which lobectomy or pneumonectomy is required. The prognosis of carcinoma and other malignant tumors of the tracheobronchial tree is not so hopeless as it was a few years ago, because of the continuing development of means of earlier diagnosis and improvement in the technic of treatment.

Tumors of the Trachea.—Various benign tumors are encountered in the trachea, for example, papilloma, lymphoblastoma, aberrant thyroid. Symptoms are wheezing, dyspnea, cough, and hemoptysis. Diagnosis is by means of the roentgen ray and endoscopic examination (See Figs 480, 481.) Treatment is endoscopic removal, using forceps and electrocoagulation (surgical diathermy). Tracheotomy may be required, but should not be done unless necessary.

Carcinoma not infrequently occurs in the trachea and produces symptoms similar to those produced by benign tumors. The diagnosis is made by tracheoscopy and biopsy. Roentgen ray examination may be of value in outlining the lesion, if it is in the upper half of the trachea, and planigraphic anteroposterior studies should be made, as well as lateral projections (Fig 482). Treatment by irradiation (protracted fractional technic) is of considerable palliative value, but rarely if ever curative. Surgical excision is impossible in most cases.

Benign Tumors of the Bronchus—**Adenoma**—Adenoma is considered by some to be potentially malignant, but in my experience little evidence has been found to support this belief. Errors in histopathologic diagnosis probably account for most of the discrepancies in the views of different authorities on the subject.

Certainly this tumor is the most important and interesting of the benign tumors encountered in the bronchi. It has also been called a "carcinoid" (Peroni), "benign glandular tumor" (Clerf and Crawford), simply a "tumor" (Stout), and "mixed tumor" (Womack and Graham). The pathologists encounter great difficulty in recognizing these lesions, and not infrequently call them carcinomas. On the other hand, they occasionally err in the other direction, mistaking a carcinoma for a benign adenoma. Some pathologists feel that these tumors are to be considered adenocarcinomas of low grade malignancy rather than benign neoplasms. The early diagnosis is of great im-



Fig 482—Planigraphic study showing carcinoma of the trachea.

portance regardless of the benign or malignant character of the lesion, because, even if benign, bronchial obstruction, with subsequent bronchiectasis, pulmonary abscess, or empyema, will result if the growth is not removed. The question of malignancy is important also, because if malignant, some of these tumors would be operable by lobectomy or pneumonectomy, and if benign, the patient should be spared the operative risk and mutilation of these procedures.

In a series of twenty of these tumors recently reported by Dr. Konzelmann and myself,^{1 2 3} 75 per cent occurred in women, and the majority of the patients were in their second or third decades of life.

PATHOLOGY—A number of the cases of adenoma in the author's series were at first called carcinomas by competent pathologists. In one case which aroused a considerable amount of discussion the section was studied by a pathologist who is regarded as a leading authority on neoplastic diseases and this man stated that he believed the tumor would eventually prove itself to be a peculiar pulmonary

metastasis indicate their relatively benign character. My experience has led me to feel that it can be definitely stated that *these tumors do not have any inherent tendency to become malignant if they ever do*. The contrary impression has in my opinion been due to confusion and error in histopathologic diagnosis.

In the typical case of benign adenoma (Fig 483) the surface is generally covered with



Fig 483.—Typical histopathologic picture of adenoma of the bronchus

cancer. The patient is still perfectly well twelve years after bronchoscopic removal of the tumor from the left bronchus. While the term bronchial adenoma, now so generally in use, does not seem entirely acceptable in view of the bizarre histologic manifestations of these tumors, certainly their cellular structure and their apparent incapacity to infiltrate or to

squamous epithelium and a wide band of connective tissue is interposed between the surface and the tumor itself. The tumor cells are generally grouped in nests, sometimes without any particular arrangement, but more often the peripheral cells are seen to form a definite layer, such as is noted characteristically in basal-cell carcinoma of the skin. The more central cells

8. Tumor of left lower lobe bronchus in a woman forty-five years of age, complaining of productive cough of five years' duration with some hemoptysis. This tumor was removed with bronchoscopic forceps and radon seeds implanted into its base. In addition a course of roentgen ray therapy was given. In two months the patient gained 30 pounds in weight and symptoms disappeared.

9. Large tumor of the main bronchus in a woman fifty-six years of age, complaining of productive cough for one and a half years with some hemoptysis. This patient had had at one time a tentative diagnosis of pulmonary tuberculosis. However, because of the signs of obstruction of a large bronchus, bronchoscopy was decided upon, and the tumor was revealed. This tumor was removed by means of forceps and electrocoagulation with a good result, but the ultimate outcome is still regarded as doubtful.

10. Tumor of the lower lobe bronchus in a woman aged twenty-eight years, complaining of productive cough intermittently for two years, dating from three attacks of pneumonia occurring within one month. Biopsy showed a glandular neoplasm, at first regarded as malignant but subsequently diagnosed as benign adenoma.

11. Tumor projecting from the left upper lobe bronchus of a patient who had been hospitalized nine months in a sanatorium under a tentative diagnosis of tuberculosis because of hemoptysis and an upper lobe atelectasis. This tumor was removed piecemeal with bronchoscopic forceps. Histologic study showed it to be a typical adenoma.

12. Smooth, reddish polypoid tumor of the left main bronchus in a woman forty-two years of age, complaining of a dry hacking cough and wheezing for one year previously. This tumor was removed piecemeal with bronchoscopic forceps.

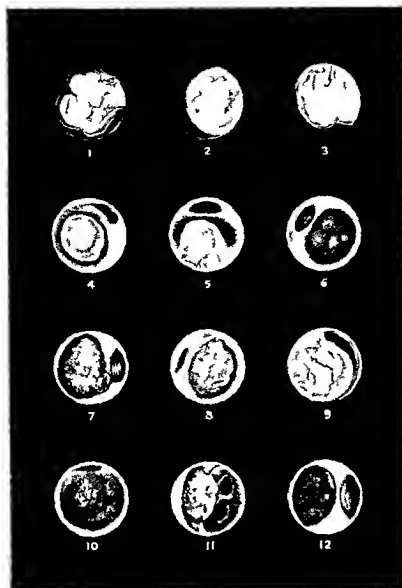


Fig 484.—Endoscopic views of bronchial adenomas.

1 Tumor of right bronchus originally reported as endothelioma this tumor was successfully removed with bronchoscopic forceps and the patient is well today twenty five years afterward

2 Tumor obstructing the right bronchus treated by bronchoscopic removal and roentgen ray therapy with the addition of transfusions because of secondary anemia resulting from bronchial hemorrhages This tumor was originally diagnosed adenocarcinoma but review of the sections leads us to regard it as a benign adenoma The patient remains well twelve years afterward

3 Pinkish tumor mass obstructing the left lower lobe bronchus This tumor like the previous one, was originally diagnosed adenocarcinoma by the pathologist but recent review of the old sections has led us to consider the lesion a typical benign adenoma

4 Tumor of the right lower lobe bronchus in a girl fifteen years of age producing atelectasis of the right lower lobe This tumor was removed with forceps but has shown a tendency to local recurrence

5 Tumor of the right lower lobe bronchus in a woman twenty five years of age which was removed with bronchoscopic forceps Histologic study showed a structure very similar to that of the preceding tumor

6 Lobulated reddish tumor of the left lower lobe bronchus in a woman thirty two years of age who complained of recurring pulmonary hemorrhages for two years prior to her coming to us This tumor was removed with bronchoscopic forceps about five years ago and has not recurred The photomicrograph shows the lesion to be a typical bronchial adenoma though several pathologists regarded the histologic picture as suspicious of malignant change

7 Large tumor of the left main bronchus causing almost complete obstruction. The roentgen ray picture was that of obstructive emphysema of the left lung Subsequently atelectasis of this lung developed Bronchoscopic removal of the tumor with forceps and electrocoagulation resulted in reiteration of the lung and the patient is now almost symptom free five years after the onset of symptoms.

Continued on preceding page

lie loosely, without any particular relationship, but sometimes a lumen is seen in the center of these cell nests. The nuclei have the structure of normal or non neoplastic cells. There is a fine but definite nuclear membrane, and a fine distribution of chromatin particles. Mitotic figures are extremely rare. The histopathology of these tumors has been discussed in detail in the recent paper by Jackson and Konzelmann.¹ Incidentally, it is probable that many of the tumors reported as "inflammatory tumors," "polyps," "hemangiomas," "fibromas," and other benign bronchial tumors are really adenomas. Several of my early cases were thought to be "tumors of inflammatory origin," but after careful his-

physical and roentgen ray examination, signs of obstructive emphysema or obstructive atelectasis may be found. Most patients present very few symptoms or physical signs in the early stages.

BRONCHOSCOPIC APPEARANCES AND BIOPSY—Benign adenomas, seen through the bronchoscope, generally appear as smooth, rounded, reddish tumors in the larger bronchi, projecting into the bronchial lumen without infiltrating the wall as the malignant tumors do. Sometimes the color is grayish rather than reddish, and not infrequently some blood stained mucopurulent secretion is present (Fig. 484). Bronchoscopic biopsy is generally easy, but as stated above, the

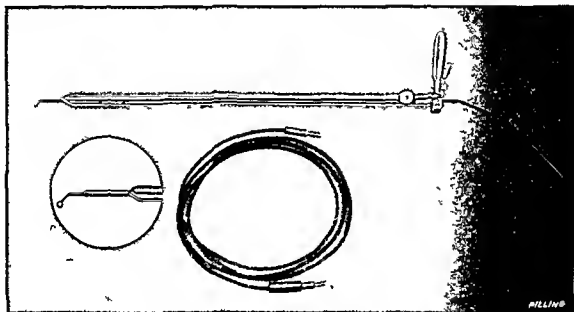


Fig. 485.—Bronchoscope and electrodes used for electrocoagulation

tologic study it was decided to group them with the adenomas. Pollak, Cohen and Gnassi published a case report several years ago, to which they appended a most interesting review of the literature and tabulation of reported cases. "Inflammatory tumors" constituted 22 per cent of the cases, but adenomas predominated, constituting 49 per cent. The entire series comprised 104 cases, twenty-seven diagnosed at autopsy and seventy seven diagnosed by bronchoscopic biopsy.

SYMPTOMS—*Hemoptysis* is perhaps the most common symptom, and for this reason many of these patients are sent to sanatoria with a diagnosis of tuberculosis. Other symptoms were those of bronchial obstruction, for example, wheezing, productive cough, and dyspnea. On

histopathologic diagnosis is difficult and careful study is required.

TREATMENT—*Bronchoscopic treatment* of benign bronchial tumors, by forceps removal, implantation of radon seeds, and electrocoagulation, has been practiced by a number of bronchoscopists and is generally successful in maintaining the patency of the bronchial lumen, though often a number of treatments are required. In the more vascular tumors electrocoagulation has a great advantage because it does not precipitate hemorrhage. The current is easily controllable, and should be maintained at a low point, sufficient to whiten without charring the tissue. A bipolar current is used, the neutral lead being attached to a plate under the patient's back and the active one to the

electrode which is passed through the bronchoscope. An ordinary bronchoscope may be used but there is definite advantage in using a bronchoscope with a special groove for the electrode as suggested by Kernan because in this way the shaft of the electrode is held out of the line of vision and besides one obtains better control of the electrode (Fig 485).

Surgical treatment by lobectomy and pneumonectomy is justifiable in some cases of bronchial adenoma but I am of the opinion

unusual vascularity making hemorrhage a great risk even with the use of electrocoagulation and (3) extensive secondary suppurative disease of the distal portion of the lung.

Other Benign Tumors of the Bronchus—
Papilloma occurs very rarely in the bronchus except as a secondary aerial implant in patients with papilloma of the larynx. *Lipoma* may occur in the bronchus but is even less common than in the larynx. *Osteochondroma* or *enchondroma* has occurred a few times in my

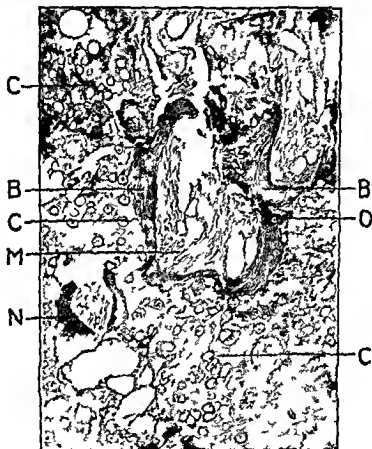


Fig 486—Sect on of osteochondroma of the bronchus B Bone C, cartilage M and N marrow cavity lined by osteoblasts O disintegrating bone surrounded by osteoclasts 3

that a trial of bronchoscopic treatment should be given in every case. If there is complicating drowned lung, bronchiectasis or abscess distal to the tumor, these conditions can be greatly benefited by the relief of obstruction and the restoration of aeration so that operative risk is greatly reduced. If these conditions are not present, very probably surgical treatment will not be required. The indications for lobectomy and pneumonectomy, as I see them, are (1) impossibility of bronchoscopic removal after a fair trial by a skilled bronchoscopist, (2)

experience (Fig 486) and a few cases have been reported in the literature. *Granulomas*, both specific and nonspecific, occur and can be removed bronchoscopically. Perhaps the most common is tuberculoma (q.v.).

Neurofibroma is a benign tumor but it extends widely to involve adjacent structures and may intrude into the bronchus. In one of our cases, surgical excision of a large tumor mass had been done by thoracotomy but obstructive atelectasis developed subsequently and diagnostic bronchoscopy showed endobronchial in-

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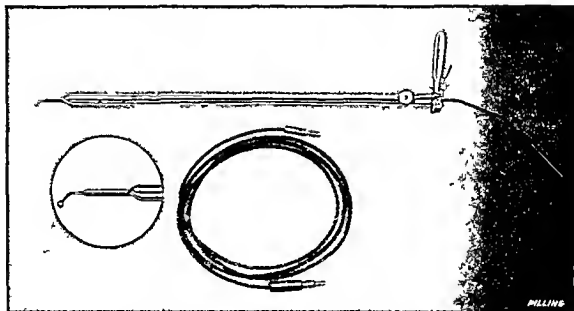


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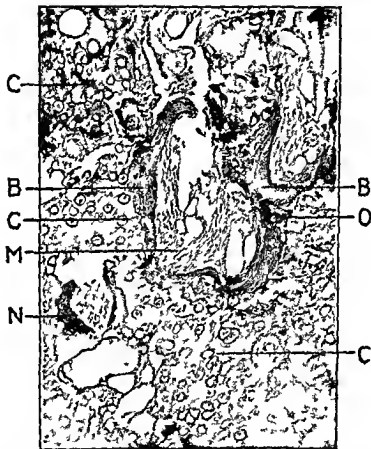


Fig 486—Section of osteochondroma of the bronchus. B Bone. C cartilage. M and N marrow cavity lined by osteoblasts. O disintegrating bone surrounded by osteoclasts.

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trusion. Bronchoscopic biopsy showed the presence of a neoplasm inside the bronchus which was histologically identical with that removed by thoracotomy (Fig 487)

diagnosis and earlier recognition, while others insist that there is a definite increase. Relative sex and age incidence in a series of 300 cases recently analyzed in our records at the Temple



Fig 487—Neurofibroma in a woman aged eighteen years. A, Before the removal of the tumor by thoracotomy. B, After the removal of the tumor by thoracotomy. C, Obstructive atelectasis of the entire right lung developing 2 years after the operation due to intrusion of the remaining portion of tumor in the bronchus with complete obstruction. D, Partial re-aeration of lung after removal of the endobronchial portion of the tumor with forceps and surgical diathermy.

Malignant Tumors of the Bronchus—*Carcinoma*—**ETIOLOGY**—Theories concerning the cause of cancer of the bronchus are interesting but none have been proven. With regard to incidence there has been much discussion, some authorities like Boyd, claiming that the apparent increased incidence is due to improved

University Clinic might be of interest. Two hundred and sixty-two, or 87 per cent, of the patients were males and thirty-eight (13 per cent) were females. One patient was under twenty years of age, and one over eighty, but almost half (126 or 42 per cent) were in the sixth decade of life.

PATHOLOGY—Primary carcinoma of the bronchus may be squamous-cell carcinoma or adenocarcinoma. Various pathologists have offered different classifications. Jackson and Konzelmann⁴ suggested a classification several years ago which is still in use, in slightly modified form, at the Temple University Hospital. Any detailed consideration of the pathology of carcinoma of the bronchus would be out of place here, but the observations relating to the mechanism and consequences of bronchial obstruction outlined in a preceding paragraph apply equally to malignant as to benign tumors.

SYMPTOMS—Cough, hemoptysis, wheezing, dyspnea, and pain are among the commonest symptoms of cancer of the bronchus. When there is complicating suppuration, cough is productive and there is fever and weight loss. Hoarseness will be produced when there is involvement of one of the recurrent laryngeal nerves.

DIAGNOSIS—Physical examination will reveal no abnormality in the early stages, but before long the signs of bronchial obstruction of some degree will be noted. Roentgen-ray examination may show a slight area of density in the early stages, or may show no changes. When bronchial obstruction develops, obstructive emphysema or obstructive atelectasis will be present. In the case of the more peripheral tumors, a mass density may be seen, but in tumors of the larger bronchi the shadow of the tumor itself is generally obscured by the density of atelectasis. Pleural effusion may likewise mask the picture.

BRONCHOSCOPIC APPEARANCES—Diagnostic bronchoscopy is important whether or not the tumor is visualized and tissue taken for biopsy study. The bronchoscopic appearance of carcinoma, when the lesion is visualized, is generally that of a nodular roughening of the surface of the bronchial wall, or of a nodular mass of tissue projecting into the bronchial lumen (Fig. 488). Proximal to the tumor itself, very often a puckering of the bronchial mucosa and some degree of stenosis are noted. The bronchus is generally less displaceable than normal, or even rigidly fixed. Widening of the carina suggests the presence of lymphoid metastasis.

BIOPSY DIAGNOSIS—In the series of 300 cases that has previously been mentioned the diagnosis was proven histologically in all cases, either by biopsy or by autopsy. Bronchoscopic

biopsy was done in 216 (72 per cent), aspiration biopsy in forty one (14 per cent). The diagnosis was made by biopsy of a node in eleven (4 per cent), at operation, either exploratory or lobectomy or pneumonectomy, in eight (3 per cent). In two cases tumor cells were found in sputum and in one case biopsy was done by thoracoscopy. Only autopsy proved the diagnosis in nine cases.

Bronchoscopic biopsy is of course to be desired, but if the lesion is not visualized in the bronchoscopically accessible bronchi, needle aspiration biopsy should be done, under fluoroscopic guidance, if the clinical evidence makes carcinoma a strong diagnostic probability. The day will probably come when exploratory thoracotomy is undertaken with as little hesitation as exploratory laparotomy, but certainly it is preferable to establish the diagnosis preoperatively if possible.

TREATMENT—The only curative treatment at the present time is resection by lobectomy or pneumonectomy so the first step after establishing the diagnosis is to evaluate the particular case with respect to operability. Operability will depend first on the presence or absence of metastasis, next on the location and extent of the lesion, and particularly on the involvement of contiguous organs. *Roentgen-ray treatment* is of very little use in the treatment of bronchial carcinoma, but its use may be justifiable in some of the operable cases.

PROGNOSIS—The prognosis of cancer of the bronchus is still not good, but it is much better than before the development of present-day methods of early diagnosis and the modern technique of anesthesia and surgery in the thorax.

Other Malignant Tumors—*Sarcoma* of the bronchus occurs much less frequently than carcinoma. Its diagnosis and treatment are similar when it does occur.

Lymphoid tumors, including *Hodgkin's disease*, may involve the bronchus by actual invasion, or by compression, though they are rarely diagnosed by bronchoscopic biopsy. Their diagnosis is more often made by roentgen-ray appearances and biopsy of lymph nodes. It is important not to mistake them for carcinoma because they are radiosensitive, and unlike carcinoma they should certainly receive roentgen-ray treatment. The dosage, however, should be light, in order to obtain the most prolonged effect and to be able to give successive courses.

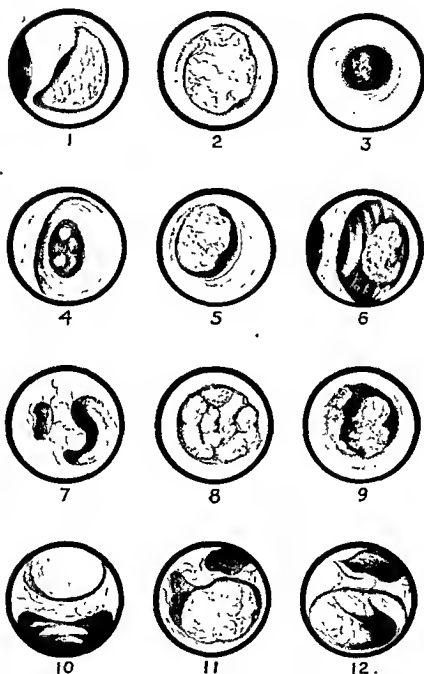


Fig. 488.—Bronchoscopic views of carcinoma of the bronchus.

1. Pedunculated tumor in right bronchus, carina thickened anteriorly.
2. Tumor of right lower-lobe bronchus (squamous carcinoma grade III).
3. Carcinoma originating in bronchial stenosis due to inflammatory consequences of prolonged sojourn of a tooth as foreign body.
4. Squamous-cell carcinoma grade III in bronchus from which a broncholith had been removed.
5. Carcinoma of "indifferent" type (grade IV) in patient who had syphilis for many years.

Secondary Malignant Tumors of the Bronchus
 —In every case of bronchial tumor, the possibility of its being secondary should be borne in mind, and appropriate studies to exclude a primary tumor in some other part of the body be carried out. Secondary tumors are a little less often accessible to bronchoscopy than primary ones, and when they are not visible bronchoscopically, needle aspiration should be done under fluoroscopic guidance, as in the case of primary tumors. *Treatment* of secondary tumors is generally of little avail. Certainly resection is not justifiable, but if the tumor is radiosensi-

tive, of course roentgen-ray treatment should be given.
 CHEVALIER L. JACKSON

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- 6 Adenocarcinoma arising from distal spur of right upper lobe bronchus in a man forty two years of age with crowding in of the lateral bronchial wall below
 - 7 Carcinoma showing itself as irregularity of spur of orifice of medial basal bronchus
 - 8 Carcinoma of "indifferent" type occluding right bronchus
 - 9 Adenocarcinoma grade III filling right bronchus with grayish yellow nodular masses springing from bloody ulcerated bed
 - 10 Metastatic carcinoma of clear cell type (hypernephroma) filling orifice of middle-lobe bronchus
 - 11 Tumor mass occluding right lower lobe bronchus, with bronchial wall rigid and deformed (squamous-cell grade III)
 - 12 Carcinoma (squamous cell grade III) obstructing the right lower lobe bronchus and also causing almost complete occlusion of the middle lobe orifice

PART VI ESOPHAGUS

APPLIED ANATOMY OF THE ESOPHAGUS

The esophagus is a muscular canal formed by the continuation downward of the inferior constrictor muscle of the pharynx. It starts from the

of the cricoid cartilage at the sixth cervical vertebra. It is 10 inches (25 cm) long and goes through the diaphragm at the tenth or eleventh thoracic vertebra, 16 inches from the teeth. It is crossed by the arch of the aorta back of the middle of the first piece of the sternum, 10

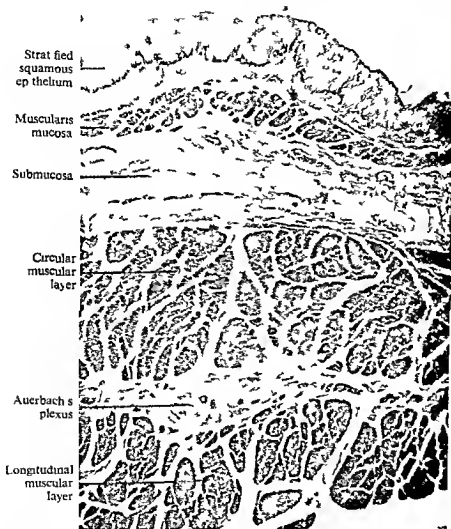


Fig. 489—Infant two years old. Sect. on from the middle of the esophagus to show normal histologic structure.

back of the cricoid cartilage opposite the sixth cervical vertebra. At the mouth of the esophagus the lower border of the inferior constrictor muscle projects like a mound into its lumen and acts as sphincter. The esophagus begins 6 inches (15.2 cm) from the incisor teeth, back

10 inches (25 cm) from the teeth. The measurements to be remembered in connection with it are then 6 and 10.

Structure.—The esophagus has an outer muscular coat of two layers and an inner glandular coat covered with stratified squamous

or pavement epithelium. A connective tissue layer joins the two. The outer layer of the muscular part consists of longitudinal fibers and the inner layer of circular ones. The thickness of the esophagus is 3 to 4 mm. The anterior longitudinal fibers are attached to the back of the cricoid cartilage on a median ridge. The inner layer of circular muscular fibers is a continuation downward of the fibers of the inferior constrictor muscle. The upper end of the esophagus is the lower end of the pharynx, so that the voluntary muscular fibers predominate. Thus it happens that a foreign body arrested at the entrance of the esophagus is often thrown back into the pharynx and into the mouth.

and the longitudinal muscles. They are found mainly in the lower half of the esophagus. Meisner's plexus is found in connection with the muscularis mucosa. A few ganglia of Auerbach's plexus are found in the upper part of the esophagus.

The upper third or half of the esophagus is composed of striated voluntary muscle and the lower half of nonstriated muscle. Some voluntary muscle is found at times around the esophageal opening of the diaphragm.

The Connective Tissue Sheath.—The esophagus is surrounded by a very abundant connective tissue sheath (Fig. 490). This is continuous without break with the lesser omentum

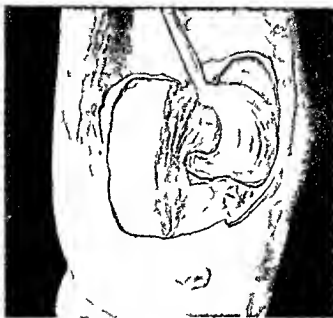


Fig. 490.—Baby dead at birth, dissected to show the relation of the lesser omentum to the stomach and to the lower end of the esophagus. Notice the thickening of the left crural angle. The writer believes that the angle between the left crus and the esophagus is the point of election for thickening of the connective tissue about the esophagus, the thickening being due to infection of the glands in this locality.

Lymphatics.—The lymphatics of the pharynx enter both the mediastinal and the cervical glands, so that in suspected cancer of the esophagus the glands at the root of the neck should be examined.

Arteries and Veins.—The arteries of the esophagus come from the thoracic aorta and are four or five in number. The veins end in part in the vena azygos major and in part in the vena azygos minor.

Nerve Supply.—The vagus is the dilator of the esophagus; the sympathetic of the constrictor. Auerbach's plexus of the sympathetic furnishes numerous large ganglia between the circular

The connective tissue is more abundant between the left crus and the esophagus. Many glands are placed in the sheath. In one specimen of the esophagus, that of a baby dead at birth, there was a great enlargement of the glands at the bifurcation of the trachea. Both trachea and esophagus were surrounded by glands at this point, and a tubular sheath of glands followed the esophagus to the esophageal opening in the diaphragm. Between the crura and the esophagus, especially between the left crus and the esophagus, there was a nest of glands suggesting a plexus similar to the one at the bifurcation of the trachea.

The Bed of the Esophagus—If the bodies of the thoracic vertebrae are wide and flat the esophagus has a secure bed, but not if they are rounded and narrow. The heart lies on the front face of the esophagus like a weight. When the esophagus becomes loosened from its bed it is dislocated nearly always to the right. I have found but three instances in which the esophagus lay to the left of the vertebral column. The esophagus is surrounded by an extensive layer of periesophageal tissue except for about 3 or 4 inches (7.6 to 9.1 cm.) at the bottom. Here the posterior surface of the esophagus is free (Brickley¹).

Movements of the Esophagus—As the front portion of the beginning of the esophagus is attached to the median ridge of the posterior surface of the body of the cricoid cartilage, in swallowing the esophagus follows the larynx upward about 2 inches (5.0 cm.).

Following the excursions of the diaphragm the lower end of the esophagus moves up and down $1\frac{1}{2}$ inches (3.81 cm.). Owing to its lack of attachment posteriorly, the terminal portion of the esophagus moves freely. On account of the twist of this part of the esophagus to the left, as the diaphragm moves upward in expiration there is a slight downward pouching of the right wall of the esophagus. Thus pouching and the twist of the terminal portion of the esophagus to the left form a perfect setting for the characteristic deformity found in fibrosis of the terminal end of the esophagus (cardiospasm). In fibrotic strictures of the esophagus below or near the lower border of the cricoid cartilage, the periodic excursion of the upper end of the esophagus plays a part in bringing undue pressure on the posterior wall where this is anatomically weak, that is, in the weak triangle of the esophagus. Thus both the movement of the upper and the lower ends of the esophagus play a part in two deformities of the esophagus—pharyngeal pouch and fibrosis of the terminal portion.

It almost seems as if Nature tried to get both ends of the esophagus into trouble and often succeeds, especially at the lower end.

Direction—The esophagus is placed for the most part a little to the left of the middle line. In the middle of its course, at the fourth thoracic vertebra, it swings to the middle line, back of the arch of the aorta, but at once goes to the left again and enters the stomach to the left and in front of the aorta at the eleventh thoracic

vertebra. This deviation from the middle line does not interfere with the passing of bougies or tubes except at the lower part where the esophagus passes through the diaphragm.

Position—The esophagus has the vertebral column behind it and the trachea in front and lying on it. At the fourth thoracic vertebra the arch of the aorta makes a transverse constriction in it and a vertebra lower down the left main bronchus, at the fifth thoracic, makes an oblique line across its front surface. Below this point the heart lies on it like a weight. In the lower part, the right and left pneumogastric nerves lie on the sides of the esophagus, and back of the arch of the aorta the thoracic duct crosses from right to left behind it on the front of the vertebral column.

The lateral lobes of the thyroid gland are in relation with the upper end of the esophagus and occasionally are prolonged so that they meet in front of it (Orton²). Below the larynx the recurrent laryngeal nerves lie between the trachea and the esophagus.

The left side of the neck is usually chosen as the point of surgical approach to the esophagus as it lies a little to the left of the median line, and so is nearer on the left side. The carotid sheath containing the great vessels of the neck has to be displaced in order to see the side of the trachea and the esophagus lying behind it. In entering the upper half of the esophagus (the procedure most commonly called for) the superior thyroid artery may be encountered. If, in order to displace the carotid sheath, a thyroid vein has to be tied and cut the ligature should not be placed too near the internal jugular vein because the tie is liable to slip. Furious bleeding results. The bleeding stump of the thyroid vein is hard to clamp, and tying the internal jugular vein itself is quicker and safer in such a mishap.

The Diameter—Only in the region of the mouth of the esophagus is the diameter relatively fixed. The esophagus is constricted at four points. Of these, the upper and the lower are the most important. The upper one is caused by the projection backward of the cricoid cartilage, the lower, by the encircling fibers of the diaphragm. The upper one hinders the introduction of the examining tube, the lower one obstructs the passage of the esophagoscope into the stomach. The first constriction is a transverse slit slightly less than 1 inch (2.54 cm.) wide, the second is about the same width. The long axis of this second constriction is from

right to left from behind forward. The lumen of the esophagus at this point is subject to wide variations, which depend upon the relaxation or the contraction of the diaphragm. Often the two less important constrictions are not seen on esophagoscopy unless watched for, and they disappear completely if large tubes are used. The first of these supplemental constrictions corresponds to the arch of the aorta and is found behind the junction of the first and second pieces of the sternum and in front of the fourth thoracic vertebra. The other constriction, which is the third from above downward, is made by the crossing of the left bronchus in front of the esophagus, and occurs at the level of the fifth thoracic vertebra. Roughly the transverse diameter of the esophagus is 1 inch at its beginning and at the lower end.

Distensibility.—All the constrictions of the esophagus are distensible. The upper constriction is less dilatable than the others so that this is the one which gives the greatest trouble in esophagoscopy. The normal esophageal wall, according to Jackson,³ will stretch 2 cm without rupture. At times, foreign bodies stretch it more than this.

The Esophageal Glands.—There are two sets of glands in the esophagus—the superficial and the deep. The superficial glands lie above the muscularis mucosa, and occur in greatest numbers in two places, first from the level of the cricoid cartilage down to the bifurcation of the trachea, and second at the terminal portion of the esophagus. The superficial glands are important because when they are found in biopsy specimens they have often led to a diagnosis of aberrant gastric mucosa. Peptic cells must be present in the glands before such a diagnosis can be made. It is characteristic of such cells that they take the eosin stain very brilliantly.

The upper set of superficial esophageal glands often cluster in quite sizable patches, and these are very prone to ulceration. One at once wonders if the lower set at the cardia has the same habit. I mention the esophageal glands for another reason. It is common to find a striking change in the character of the last half inch of the esophageal mucosa. There is often a ringlike band which stands out distinctly from the mucosa of the esophagus and from that of the stomach. It is generally of a bluish color. Histological examination shows many glands. I take it, therefore, that their presence

accounts for the change in the character of the mucosa at this point.⁴

Lymphatic Glands.—In examining the lower end of the esophagus in children, I have often found very sizable glands placed between the crura and the esophagus (Fig. 491). The same is true of specimens of the adult esophagus. The presence of these glands is important be-

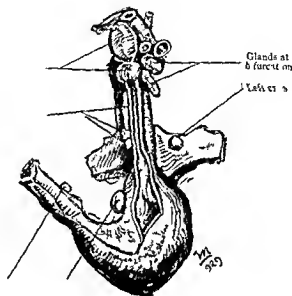


Fig. 491.—The esophagus and stomach of a baby dead at birth. The purpose of the illustration is to show the position of the lymphatic glands about the esophagus. The glandular enlargement in this specimen is typical of that found in tuberculosis. There is a large mass of glands at the bifurcation of the trachea which surrounds both the trachea and the esophagus. There is a glandular chain on both sides of the esophagus down to the diaphragm. This is more marked on the right. There is a good sized gland between the left crus and the esophagus. Other specimens show that this gland is very constant. Below the diaphragm on the right and at the top of the lesser curvature of the stomach there is another large gland. This also is generally found. The illustration shows a small gland at the pylorus of the stomach.

cause they act as the middleman in carrying infection to the esophagus.

The Subdiaphragmatic Esophagus.—Dissection shows that there often is no subdiaphragmatic esophagus. In such cases the edges of the crura lie on the stomach itself. The anterior crus is thin and may even be membranous. The posterior or left crus is sturdy and makes a marked crease on the posterior surface of the

esophagus and notches its left border (See Figs 492 497 498)

The Cone of the Diaphragm—The two halves of the diaphragm meet in the midline and in front of the spine dip down in a cone the tip of which is made by the crura Through this cone runs the terminal part of the esophagus and

the cone of the diaphragm opens and flattens out When this happens the terminal part of the esophagus is set afloat The esophagus bobs about aimlessly on the flattened diaphragm and turns and twists as it pleases

The Crural Sling—The form of the sling made by the crura varies and with it the form



Fig 492 The subdiaphragmatic esophagus Drawing from a dissection of an adult cadaver to show the relation of the left crus to the esophagus and to the fundus of the stomach Notice that the left crus lies on the fundus of the stomach and that there is no esophagus below it The right crus has been removed Its left edge was parallel and flush with the left edge of the left or posterior crus In this subject therefore there is no subdiaphragmatic esophagus In six subjects three others were found in which the crura had the same relationship as the one pictured Thus it appears that the subdiaphragmatic esophagus is not constant

the cone intervenes between the esophagus and the liver The cone of the diaphragm is packed with loose connective tissue It binds the esophagus more firmly on the front and sides than behind The cone of the diaphragm acts as a sleeve which keeps the terminal part of the esophagus in line With falling of the diaphragm

of the terminal portion of the esophagus The amount of support which the sling gives to the esophagus varies with the width of the crural angle Normally the two crura come together sharply before they overlap and they cover a very considerable portion of the posterior surface of the esophagus In some cases however

the angle is wide and almost no support is afforded by them

In the dilated sagging esophagus such as we find in late cases of fibrosis of the esophagus the wide open crural sling conceivably may have a clothesline effect and could easily play a part in the backward bend which is so important a factor in the obstruction present in these cases

The Crura and the Esophagus at Birth—Fortune favored me when I tried to make a frozen section study of the esophagus of a baby dead at birth. I made a transverse vertical cut

anteroposterior and that the terminal portion of the esophagus the oblique or crural arm is covered by the left crus. A metal cast shows that the crural or oblique arm is a thin anteroposterior ribbon. The barium picture of the lower end of the esophagus has puzzled me for years. I feel now that it is reasonably clear, thanks to the baby (Figs 493, 494, 495).

The Liver Tunnel—The terminal part of the esophagus is not only surrounded by the cone of the diaphragm but it passes through a tunnel of surrounding liver and makes a distinct impression on the posterior surface of the liver

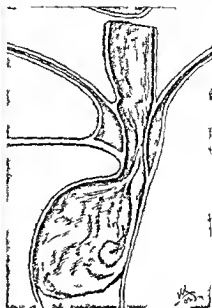


Fig 493 —Metal casts from babies dead at birth. A The first cast shows that the esophagus comes to a point at the edge of the left crus and changes to a thin ribbon the axis of which is anteroposterior. B The second illustration is that of a cast from another baby. The cast is in place. Notice the thickness and the crescentic shape of the left crus and the thinness of the right crus. How the crura bound and determine the shape of the crural arm is shown clearly. These casts clear up the barium picture of the terminal portion of the esophagus. The point of physiological closure of the esophagus on inspiration is where the esophagus changes its axis and direction, namely at the edge of the left crus. The marked gap in the esophagus which the roentgen ray film so often shows below the point of closure represents the width of the crura.

through the lower half of the esophagus and the crura and the stomach (Figs 493, 494, 495). Years ago I pointed out from a study of frozen sections of the adult esophagus that the esophagus comes to a point in the cone of the diaphragm and twists on itself from right to left. This led to the academic division of the terminal portion of the esophagus into a vertical arm and an oblique or horizontal arm. Frozen sections of this baby show that the esophagus comes to a point where it meets the upper edge of the left crus; that the diameter of the esophagus changes here from transverse to

Between the esophagus and the liver the cone of the diaphragm and the crura which make the point of the cone, of course, intervene.

The liver is chiefly responsible for the shape of the lower end of the esophagus. According to the closeness of the investing liver the terminal part of the esophagus is either trumpet shaped or cone-shaped. The lower end of the esophagus has the liver on the right in front, and in many cases a thin tongue of liver hooks round its left edge like a sickle. Behind the esophagus is the descending aorta which separates it from the vertebral column. The liver tunnel as studied

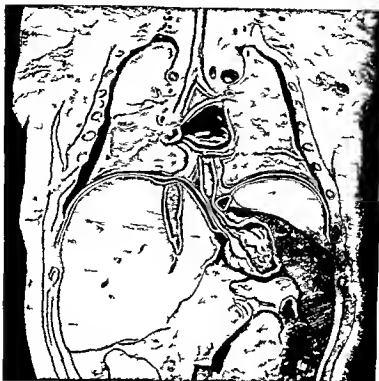


Fig 494 —Photograph of a frozen sect on from a baby dead at b rth The cut s through the terminal port on of the esophagus and through the stomach The terminal port on of the esophagus is seen to come to a point at the top of the left crus (shown by dark triangle adjacent to the esophagus) The esophagus then changes its axis from transverse to anteroposterior and becomes a thin ribbon lying between the crura Notice the intimate relation of the left lung tip to the esophagus and the left crus



Fig 495 —Photograph of a frozen sect on of a baby dead at b rth The cut passes through the terminal port on of the esophagus and through both crura and the stomach The sect on is seen from behind In the stomach and the crural arm of the terminal port on of the esophagus there s a metal cast The left crus has been cut to show the cast Both crura show distinctly The esophagus s seen to come to a point at the edge of the left crus The crural arm begins at this point and runs at an angle of 45 degrees to the stomach The length of the crural arm is the width of the left crus Notice the crescentic enlargement of the left crus above the beginning of the crural arm Notice the relation of the left lung tip to the esophagus and to the left crus In this specimen the right crus is not in intimate relation with the esophagus

on the cadaver, varies greatly in length in different subjects. Owing to the extreme size of the liver at birth the liver tunnel in the baby is much longer and wider than in the adult.

The Lower End of the Esophagus As Seen in Frozen Sections.—In frozen sections the esophagus is seen to end in a point or in a slit in the middle of the diaphragmatic cone. The slit has the position characteristic of the hiatus, that is, it slants upward and to the left. When the esophagus ends in a round opening instead of a slit, the opening is to the right of the center.

A Reconstruction of the Lower End of the Esophagus from Frozen Sections.—This shows that the terminal portion consists of a vertical part and a horizontal part. Where the two arms join, the esophagus twists on itself and turns to the left. Where the turn comes the esophagus may even actually kink on itself. And where the turn comes the esophagus ends in a point. The left crus crosses behind the esophagus on a slant, running from the left above to the right below. The slanting edge of the left crus mounds into the esophagus and makes the chief boundary of the slit usually called the "hiatus" (See Figs 497, 498.)

The Horizontal Arm of the Terminal Part of the Esophagus.—This is imprisoned between the left crus behind and the edge of the posterior surface of the left lobe of the liver in front. The right crus comes in between the liver and the horizontal arm in front, but the right crus is thin and does not leave its impression on the esophagus as does the left crus. Frozen sections show that the horizontal arm of the esophagus is flat and practically closed. Backward pressure on the liver will actually close the lumen of the horizontal arm, whereas pulling the liver forward or downward opens it. Tipping the liver forward and its posterior surface backward also closes the horizontal arm. As we study the anatomy of the esophagus on the cadaver, the patency of the horizontal arm is seemingly controlled by the position of the liver.

In frozen sections, when the esophagus is taken out of the diaphragmatic cone, a large and roughly oval hole is left in which the left crus and the aorta figure prominently posteriorly, the thin right crus anteriorly. Between the anterior edge of the left crus and the left edge of the anterior crus there is a membranous area through which the horizontal arm of the

esophagus reaches the stomach. This membranous area is overlain by the liver, between the right edge of the aorta and the posterior surface of the right or anterior crus there is another membranous area. It is only when the esophagus is put back in place and resumes its natural bend to the left that anything approaching a slitlike hiatus occurs in the lumen of the esophagus.

The Terminal Portion of the Esophagus in the Adult.—The terminal portion of the esophagus in the resting state I believe to be closed on



Fig. 496.—Drawing of a reconstruction of the lower end of the esophagus (in an adult) from frozen sections. The drawing shows that the terminal portion of the esophagus consists of a vertical and a horizontal portion. The vertical part comes to a point and its axis changes from transverse to anteroposterior. The horizontal portion or arm is very much pleated. This part the writer believes to be closed in the resting condition. The closure is due mostly to the pleats (See Figs 499 and 500.)

account of the pressure of the crural canal, the presence of the deep, vertical folds and of the natural twist to the left (Figs 499, 500, 501).

Sphincters of the Esophagus.—The lower edge of the inferior constrictor muscle of the pharynx has been given a separate name, the cricopharyngeus, because it rises from the posterior lateral half of the cricoid cartilage and encircles the posterior half of the pharynx. The cricothyroid muscle is beneath it so that it does not rise from the cricoid cartilage itself but from the fascia of the cricothyroid muscle which it overlaps. Anteriorly and superiorly its

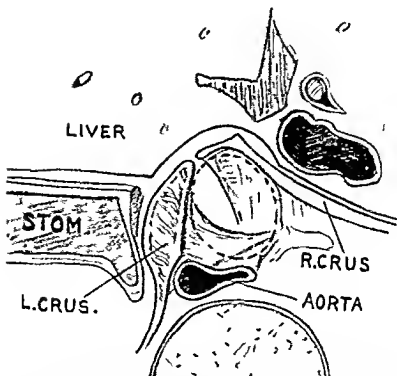


Fig 497 —Drawing from a frozen section of an adult cadaver The terminal portion of the esophagus has been removed in order to show the crura The dotted line indicates the esophagus Notice how much thicker the posterior crus is than the anterior crus The drawing shows how the left crus passes obliquely behind the esophagus and between the esophagus and the aorta The posterior surface of the left lobe of the liver is seen overlying the anterior surfaces of both crura

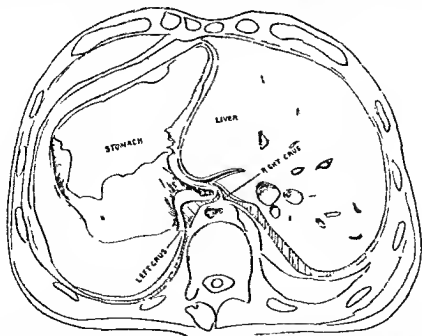


Fig 498 —Drawing from a frozen section of an adult cadaver The section passes through the horizontal arm of the terminal portion of the esophagus Notice that the left or posterior crus lies behind the horizontal arm and that the posterior surface of the left lobe of the liver lies in front The anterior or right crus in this specimen is not in relation with the anterior surface of the horizontal arm The drawing shows how backward pressure from the left lobe of the liver will close the horizontal arm



Fig 499 --Generalized arteriosclerosis in a man sixty one years of age. Specimen showing the lower portion of the esophagus and a section of the descending aorta. The second figure of the upper row depicts the aorta laid open to show patches of arteriosclerosis. The esophagus in the first figure is seen to be a narrow closed tube. The closure is due to the vertical pleats. In 2 and 3 are shown the character of the pleating at lower levels. In 3 there are two deep vertical pleats, one on the anterior wall of the esophagus, the other on the posterior wall. Notice also how these two pleats practically close the esophagus. The writer believes that this is the normal resting condition of the terminal or pleated portion of the esophagus.

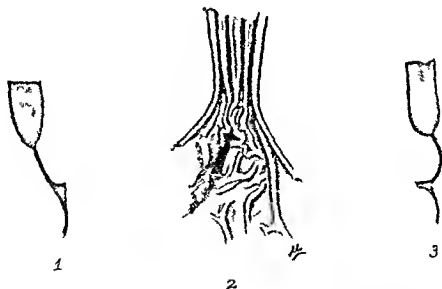


Fig 500 --The esophagus of a child. The illustration demonstrates the vertical pleats at the lower end of the esophagus. These pleats are continuous for a short distance with the rugae of the stomach. On the anterior wall of the esophagus one or two pleats are continuous with long rugae which skirt the lesser curvature of the stomach. One of these rugae is shown in the illustration. In the resting or collapsed state of the terminal portion of the esophagus the barium runs in the grooves made by the pleats. This accounts for the pecters given by the barium meal. In 1 and 3 the esophagus is seen to come to a point. Below this there is a narrow ribbon of barium. This represents a channel between the pleats in the closed esophagus. Where the esophagus ends and the stomach mucosa begins there is a slight ridge. Examination shows that this is due to a mounding of the mucous membrane of the esophagus. Although to the naked eye the esophageal mucous membrane seems to stop at this ridge, microscopical examinations show that it may extend fully 5 cm. below this point.

facial attachment begins at the tubercle at the lower end of the oblique line of the thyroid cartilage, and its attachment extends to the lower border of the cricoid cartilage. This facial origin has the shape of an arcuate ligament. A similar ligament occurs where the diaphragm crosses the psoas muscle and the quadratus lumborum in the abdomen. This tendinous arching origin of the cricopharyngeus plus the fact that it overlies the rounded cricothyroid muscle is the best explanation the writer knows for the clinical entity called the "cricopharyngeal fold." In examining the entrance of the

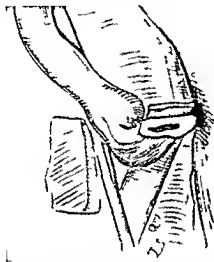


Fig 501.—Drawing of the terminal port on of the esophagus of an adult cadaver. The esophagus was not injected. The specimen shows the twist of the terminal portion of the esophagus. Notice how the left crus lies behind the horizontal arm. The anterior crus has been mostly cut away. Above the edge of the left crus there is a globular dilatation of the esophagus. When the esophagus is distended the horizontal arm straightens out and the distinction between the vertical and the horizontal arms of the terminal portion of the esophagus is lost.

esophagus especially with small tubes this fold is encountered and the distal end of the tube has to be raised a little and made to jump over it.

The cricopharyngeus encircles only the posterior half of the mouth of the esophagus. Its lower fibers, however, are continuous with the circular fibers of the esophagus so that the two make a complete sphincter about the beginning of the esophagus.

The sphincter action of the circular fibers extends a short distance down the upper part of the esophagus. This accounts for the fact that foreign bodies, especially coins, are often held

just below the cricopharyngeal fold. A coin held here is hidden by the fold and a small esophagoscope can easily pass over the coin without its being seen or dislodged. In order to see the coin it is necessary to raise the shaft of the examining tube sharply so that the distal end points toward the vertebral column. Years ago the above procedure turned a coin case of mine (the patient was the daughter of a physician who insisted on being present at the examination) from what at first was an embarrassing failure into a final success.

At the lower end of the esophagus a weak sphincter can be demonstrated occasionally but it is not constantly present.

Opposite the left crus the circular muscular fibers are thicker in very many instances than the corresponding fibers opposite the right crus. If one cares to, one can say that there is commonly a partial sphincter at the level of the crura and between them. Beginning at the line where the esophageal mucous membrane changes into stomach mucosa there is often a thickening of both muscular layers of the esophageal wall making what seems to me a perfectly good cardiac sphincter.

The cardiac sphincter is at times a definite and satisfying structure in the adult. It is not, however, always present. For instance in five out of seven specimens from adults the esophageal muscles came to a definite pointlike ending at the level of the crura and did not reach the cardia.

The musculature of the lower end of the esophagus or of the upper end of the stomach has a weak sphincter action, regardless of the presence or absence of an anatomic sphincter. Cannon⁵ made the observation that when the stomach is filled with a neutral solution this solution runs back and forth between the esophagus and stomach without hindrance. When however the solution is made acid it is shut off from the esophagus and retained in the stomach. He believes, therefore, that there is an acid control of the cardiac sphincter, the nerve channel being through the cardiac plexus (Fig 501).

The Fluoroscopic Picture in Swallowing Barium Milk.—If peristalsis is present the esophagus looks like a string of starchy, poorly formed sausages. In cases of cardiospasm the barium drops through fluid the level of which is usually at the arch of the aorta. If the obstruction is of moderate degree the barium



Fig. 502 —The illustration shows the lower end of an adult esophagus. Notice that there is a well marked cardiac sphincter.



Fig. 503 —Photograph of a specimen of the lower end of an adult esophagus. The esophageal muscles diminish in size almost to a point well above the beginning of the epithelium of the stomach. There is no cardiac sphincter.

facial attachment begins at the tubercle at the lower end of the oblique line of the thyroid cartilage, and its attachment extends to the lower border of the cricoid cartilage. This facial origin has the shape of an arcuate ligament. A similar ligament occurs where the diaphragm crosses the psoas muscle and the quadratus lumborum in the abdomen. This tendinous arching origin of the cricopharynx plus the fact that it overlies the rounded cricothyroid muscle is the best explanation the writer knows for the clinical entity called the "cricopharyngeal fold." In examining the entrance of the

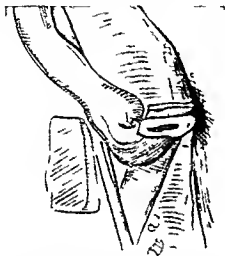


Fig 501—Drawing of the terminal portion of the esophagus of an adult cadaver. The esophagus was not injected. The specimen shows the twist of the terminal portion of the esophagus. Notice how the left crus lies behind the horizontal arm. The anterior crus has been mostly cut away. Above the edge of the left crus there is a globular dilatation of the esophagus. When the esophagus is distended the horizontal arm straightens out and the distinction between the vertical and the horizontal arms of the terminal portion of the esophagus is lost.

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two esophageal segments if possible or closure of the fistula, exteriorization of the upper segment in the neck, and anterior gastrostomy. Gastrostomy alone is not adequate. If the patient survives these procedures a new esophagus ultimately can be reconstructed.

Dilatation of the Esophagus—Dilatation of the esophagus suggesting *cardiospasm* has been observed in the newborn. Various theories concerning its origin have been suggested. The views held by Mosher regarding infection of the esophagus with fibrosis are tenable. The common symptom is regurgitation. Roentgen ray examination employing a barium mixture reveals a dilated esophagus with obstruction at

CONGENITAL SHORT ESOPHAGUS

Congenital short esophagus probably is more common than reports would indicate. Being compatible with life, cases often are not recognized early or are not differentiated from acquired stenosis. In 1931, Findlay and Kelly first described an anomaly that was characterized by a congenital short esophagus, the presence of a portion of stomach in the thoracic cavity, and stenosis at the junction of the esophagus and stomach. Opinions differ regarding the development of this anomaly. Some observers believe that the stomach is in the thorax because of hypoplasia of the esophagus which



Fig 505—Roentgenograms made in a child aged six days after ingestion of barium mixture showed a greatly dilated esophagus. Barium was observed to enter the stomach slowly through a narrowed esophageal hiatus. Considerable barium still remained in the esophagus after thirty minutes. Although the lower end of the esophagus did not exhibit the characteristic appearances of *cardiospasm*, on the roentgenograms there were observed fluoroscopically. At esophagoscopy there was no obstruction found at the esophageal hiatus and dysphagia was promptly relieved.

the hiatal level. Passage of an esophagoscope usually gives complete relief (Fig 505).

Webs—Webs producing stenosis or atresia have been reported and successfully treated by esophagoscopic perforation.

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ceased to grow early in embryonic life. Others consider the underlying factors as being identical with those causing hiatal hernia. This condition has been observed in a child, age four weeks, and its occurrence in a large number of children under ten years of age has been reported, suggesting that it is a distinct entity and should not be confused with hiatal hernia which usually is observed in middle life.

The *symptoms* are those commonly observed in cases of acquired stenosis, together with disturbances in growth and nutrition. Dysphagia and regurgitation occur when the child begins to take solid food. A careful study of the history, however, often will reveal that regurgitation has occurred since birth and that disturbances in nutrition and growth have been present

throughout the patient's entire lifetime. In other cases dysphagia, lodgment of food, and regurgitation are not marked but epigastric distress particularly after eating is a prominent symptom.

The *diagnosis* is made by roentgen ray study of the swallowing function and esophagoscopy. Many of the cases probably are considered as those of acquired stenosis. A correct diagnosis may be overlooked because of lack of proper technic in the roentgen ray study and failure on the part of the esophagoscopist to investi-



Fig 506—Roentgenogram of a child aged nine years, after ingestion of a barium mixture revealed a portion of stomach above the diaphragm, a short esophagus, and marked narrowing at the esophago-gastric junction. There is moderate dilatation of the esophagus. Rugal markings can be seen at and above the esophageal hiatus which is greatly dilated. This patient had dysphagia with frequent regurgitation since birth and exhibited marked nutritional disturbances. Esophagoscopic dilatation of the stenosis gave marked relief.

gate the food passageway distal to the stenosis and to note the level of transition of esophageal into gastric mucosa.

The essential points in the roentgen ray diagnosis of congenital short esophagus are first a portion of the cardiac end of the stomach must be shown to stay above the level of the diaphragm and, second, the esophagus must be shown to be too short to reach as low as the level of the diaphragm (Fig 506). These appearances are not unlike those noted in hiatal hernia. There is lacking, however, the tortuosity and redundancy of the esophagus observed in hiatal hernia and there is present marked stenosis at the esophago-gastric junction.

Esophagoscopy reveals a funnel like narrow-

ing of the suprastenotic esophagus which is the seat of a chronic esophagitis and exhibits some dilatation. The stenosis is firm and offers resistance to the tip of the esophagoscope suggesting an acquired stricture. There are lacking the cicatricial appearances and the eccentrically placed lumen which commonly are observed in cicatricial stenosis. Small areas of ulceration at the point of stenosis are not uncommon. These appear superficial and are covered with thin grayish exudate. As a rule, dilatation of the stenotic area can be successfully carried out to permit inspection of the substenotic portion of the food passageway. As soon as the stenotic portion is traversed one meets up with gastric mucosa at a point well above the level of the diaphragm.

The *prognosis* is good if appropriate dilatation can be carried out. Defective dental development and disturbed growth and nutrition improve with an adequate and appropriate dietary. Surgical interference commonly is not required.

The chief problems in *treatment* are providing an adequate food supply and relieving subjective symptoms. In cases of marked stenosis which resist prompt dilatation, gastrostomy may be necessary. As a rule one can secure prompt dilatation of the stenotic area by either esophagoscopic bougienage or the passage of olive tipped bougies over a previously swallowed thread. Ulceration commonly heals as soon as the stricture is sufficiently dilated to prevent stagnation of food with esophagitis. The topical application of silver nitrate (in 10 per cent solution) esophagoscopically and the employment of various combinations of alkalis afford temporary relief. The surgical problem is usually one of reconstruction and elevation of the diaphragm to a position above the stomach. This often requires interruption of the left phrenic nerve.

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HIATAL HERNIA

Improvement in diagnostic methods, particularly by roentgen-ray study and esophagoscopy examination of patients presenting obscure esophageal symptoms, and increasing success of surgical treatment are responsible for the recognition of an increasing number of cases of hiatal hernia of the stomach.

Most observers consider all esophageal hiatal hernias as essentially congenital in origin in that there is a congenital malformation of the hiatus and of its attachments to the lower end of the esophagus and stomach. Although injury may

lower end is elevated, being detached from the diaphragm, and a portion of stomach has herniated through the hiatal orifice into the posterior mediastinum. In the latter, the esophagus appears redundant, tortuous, and dilated.

Hiatal hernia commonly does not produce symptoms until adult life. A majority of the patients are over fifty years of age. The sex incidence is about equally divided.

The symptoms of hiatal hernia are complex and often are unrecognized for many years so that patients may be treated for other disorders and often are operated upon for supposed biliary, gastric, or appendiceal disease. The

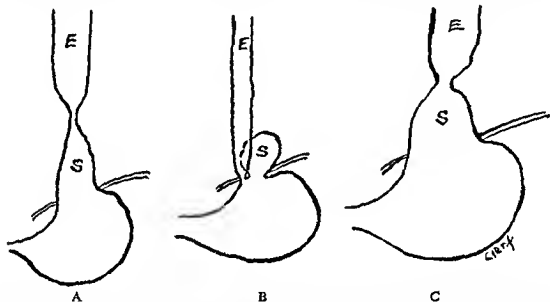


Fig 507—Drawings showing relation of esophagus (E) and herniated portion of stomach (S) to each other and to the diaphragm in (A) congenital short esophagus and in hiatal hernia (B and C). In A, the esophagus being short, terminates above the diaphragm, a portion of stomach is in the thorax and there is narrowing at the esophago-gastric junction. In B, the lower end of the esophagus remains attached to the diaphragm and a portion of stomach herniates through the hiatal opening into the posterior mediastinum. This often is referred to as a para-esophageal hiatal hernia. The esophagoscopy findings are normal. In C, the attachments of the esophagus are loosened from the diaphragm, the esophagus appears shortened, dilated, and tortuous and a portion of the stomach is herniated into the thoracic cavity.

be an exciting factor it is believed that increased abdominal pressure together with degenerative changes in the fibers encircling the esophageal opening make possible the passage of part of the stomach and occasionally other abdominal viscera into the thoracic cavity.

There are two types of true hiatal hernia (Fig 507). In one, the esophagus is of normal length and the lower end is not elevated above the diaphragm, it remains fixed in its normal position with herniation of a portion of stomach, through the ring surrounding the esophagus, constituting a gastric hernia. In the other type, the esophagus is of normal length but the

symptoms therefore may simulate such conditions as gastric ulcer, gastritis, gastric carcinoma, duodenal ulcer, pylorospasm, cholecystitis, cardiospasm, stricture or carcinoma of the esophagus, and cardiovascular conditions such as coronary occlusion, angina pectoris, and myocardial insufficiency. Frequent complaints are dysphagia, epigastric pain, gaseous eructations, pyrosis, and vomiting.

Dysphagia commonly is for coarse foods, particularly meat. The epigastric distress usually projects through the back, where it can be accurately localized and often may be referred to the left shoulder and down the arm simu-

lating angina pectoris. The symptoms are intermittent but usually are progressive. The pain commonly comes on shortly after eating or on



Fig 508 With the esophagus well filled with barium mixture and the patient in Manges position it was possible to demonstrate a small reducible hiatal hernia which had been repeatedly overlooked in routine studies of the esophagus. Esophagoscopy examination revealed marked relaxation of the esophageal hiatus. The symptoms of indigestion were relieved by appropriate medical treatment.

assuming a recumbent posture but the attacks vary in intensity depending upon the amount of stomach fixed in the hernial sac. Relief often



Fig 509—Roentgenograms show a large hiatal hernia partially filled with barium mixture. The rugal markings are well shown at the hiatal level and in the hernial sac.

can be secured by belching and vomiting or by taking alkalis. So long as the stomach is not fixed in the sac but can be reduced there may be long intervals between attacks. Symptoms com-

monly are aggravated when the patient assumes a recumbent posture. Patients may find it necessary to sleep in a sitting posture to secure relief.

The diagnosis usually can be made by roentgen ray study of the esophagus. It is necessary to use adequate opaque mixture so that the esophagus and the stomach can be outlined (Fig 508). This can be aided by placing the patient in the "Manges" or Trendelenburg's position. The diagnosis is dependent on changes in the contour of the stomach with dilatation of the hiatus oesophageus, demonstration of rugal markings, and protrusion of a portion of the stomach through the hiatus and above the



Fig 510—Roentgenogram of a large hiatal hernia shows marked dilatation and some redundancy of the esophagus with narrowing at the esophagogastric junction. At esophagoscopy the redundancy of folds produced considerable obstruction and accounted for the marked dysphagia.

diaphragm (Fig 509). Often there is noted a contraction somewhere above the diaphragm corresponding to the esophagogastric junction (Fig 510). Occasionally this may be the site of a definite cicatricial stenosis.

Esophagoscopy investigation is important not only to corroborate the diagnosis but to rule out carcinoma and also to ascertain if ulceration and stenosis are present. The esophagus is dilated and there is redundancy of mucosa. The redundant mucosa often interferes with a satisfactory inspection of the esophageal walls but this may readily be overcome by inflation. This is accomplished by placing a window plug in the proximal end of the esophago-

scope and insufflating air into the esophagus through the aspirating canal with a band bulb. This procedure also permits better visualization of the lumen at the esophagogastric junction as well as the interior of the thoracic portion of stomach. Chronic esophagitis often is present. Ulceration at the esophagogastric junction is common and may consist of small localized areas of superficial erosions covered with grayish exudate and surrounded by a narrow inflammatory zone, or it may involve the entire circumference of the food passageway. As a rule the ulceration is superficial. In questionable cases, biopsy is indicated. In the absence of stenosis no difficulty is encountered in inspecting the substenotic portion of the passageway. Long-continued ulceration commonly is followed by the development of cicatricial stenosis which may be extensive.

The *prognosis* is unfavorable. There is a tendency towards progression of the disease with fixation of the stomach and progressive interference with swallowing. Ulceration is common and when present is associated with considerable epigastric distress. Long-continued ulceration may result in cicatricial stenosis.

Treatment can be divided into medical, mechanical, and surgical. The medical treatment consists of regulation of diet, reduction of weight, and the employment of alkaline powders. This often will give complete relief. Heavy meals should be avoided in the evening. Posture is an important aid in the relief of pain particularly after eating. It may be necessary for the patient to sleep in a sitting posture to prevent gastric juices from gravitating upward into the esophagus. As a rule, however, the condition is progressive and other measures must be employed. If no benefit is derived surgical treatment is indicated.

The presence of ulceration and the development of stricture at the esophagogastric junction necessitate mechanical or esophagoscopic treatment. Topical application of silver nitrate solution (10 per cent) to the superficially ulcerated areas is of value and often gives prolonged relief. For stricture, esophagoscopic bouinage or passage of an olive-tipped bougie over a previously-swallowed thread is indicated. This often must be repeated at intervals of two or three months and continued indefinitely if surgical treatment is contraindicated.

Surgical treatment consists of the reduction and repair of the hernia without or with inter-

ruption of the phrenic nerve. Since the disease tends to be progressive and medical and mechanical treatment is palliative, surgical treatment should be recommended if the patient's condition is satisfactory. If operation is contraindicated, a rigid medical regimen should be strictly followed to prevent the distressing, often disabling, symptoms that may develop.

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FIBROSIS OF THE ESOPHAGUS

Etiology and Pathology.—It has been shown by macroscopic and microscopic examination of 100 autopsy specimens that the esophagus, like all other organs of the body, is subject to acute and chronic infection in acute and chronic systemic disease.¹ It can be infected before birth and is also subject to extensive hemorrhage at birth. The most logical explanation for so-called spasm of the pylorus at birth is a light fibrosis due to infection. The three instances, seen by the writer, in which at birth there was a narrowing of both the pylorus and the cardiac end of the esophagus, are best explained by fibrosis due to antenatal infection rather than by double spasm.

Infection of the esophagus can originate within or without the esophagus or from the blood stream by an infectious thrombophlebitis. When the esophagus is infected from within, the pathway is probably the esophageal glands. It was shown by other writers many years ago that the superficial glands at the upper end of the esophagus ulcerate easily. Infection can reach these glands readily from the pharynx, a part of the body often subjected to infection, or from the apices of the lungs. Below, the chief source of infection is disease of

the liver and the gallbladder. Autopsy specimens show that even infection of the appendix can be associated with infection of the esophagus. Periesophageal abscess is not uncommon especially at the cardiac end of the esophagus. In the last ten autopsy specimens of the esophagus which I examined, three such abscesses were found.

Dilatation of the superficial veins of the esophagus is very common. Common also is hemorrhage about these veins. I have found

portion of the esophagus has this relatively slight pathologic condition as its cause. Such cases are most dramatic because the gluing is readily broken by instrumentation, and a quick cure of the patient results. Infection followed by fibrosis of connective tissue is an old pathologic entity, and is a logical, a proved, and a common cause of stricture of all grades in any part of the esophagus.

During the past ten years Dr. Macmillan, roentgenologist at the Massachusetts Eye and

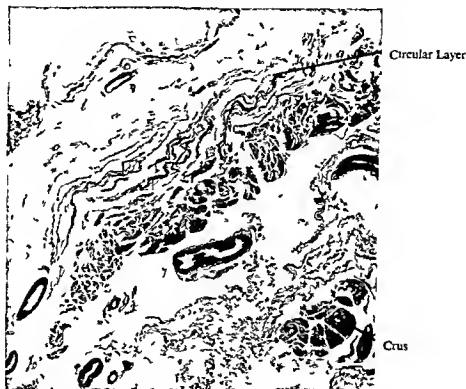


Fig. 511.—Fibrosis of the terminal portion of the esophagus in a man seventy years of age. Microphotograph of a section taken from the terminal portion of the esophagus. Mallory stain. The esophagus is completely disorganized. What is left of the circular layer is mostly fibrous tissue. There is a great increase of periesophageal connective tissue. Some muscle bundles of the crura show in this. The stain shows that they are beginning to be replaced by fibrous tissue. (See Fig. 517.)

instances in which a single vein was greatly enlarged and thrombosed and was in close contact with the esophageal epithelium. One could easily see that any further enlargement of such a thrombosed vein would tend to push off the epithelium and expose the subepithelial tissue of the esophagus to infection. In the resting state the lower part of the esophagus is closed by the deep vertical folds characteristic of this locality. An ulcer caused by trauma or infection could easily glue these folds together and result in obstruction. One type of fibrosis of the terminal

Esophagus Infirmary, has been finding more cases of fibrosis of the upper end of the esophagus than of the lower end. The fibrosis shows as a partial or complete annular stricture just below the cricoid cartilage. There is no history of the swallowing of caustics or trauma. The infection which causes the fibrosis, I believe, comes from the pharynx or the apices of the lungs, mostly from the pharynx.

The English school has held that fibrosis of Auerbach's plexus and the consequent loss of ganglion cells was the cause of the condition.

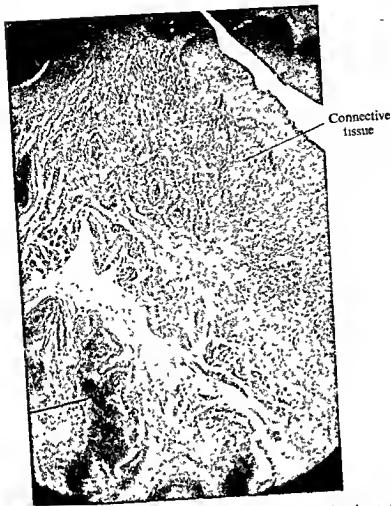


Fig. 512.—The section shows great thickening of the subepithelial connective tissue. (See marker.)



Fig. 513.—The illustration shows a large thrombosis of a superficial vessel of the esophagus. Further growth of the thrombus would push off the epithelium of the esophagus and produce a superficial erosion which could readily cause an adhesion or become an infected ulcer.

known so long as "cardiospasm" I have held that fibrosis of the plexus was a terminal condition, that it was natural in a disorganized esophagus such as is present in long-standing cases of fibrosis of the terminal portion of the esophagus. Nerve tissue has long been known to be resistant to infection. In 100 specimens, to which reference has previously been made, I found only one or two instances in which there was evidence of infection of Auerbach's plexus. I have found infection of the nerve tissue to be rare even in cases in which death was due to the most violent chest infections, for instance,

tion—of the terminal portion of the esophagus. This bend causes the quick return of obstruction after partial dilatation by instrumentation. Patients suffering from cardiospasm who complain of much pain I believe to have ulcer as the cause of the condition or as a complication.

In cases of fibrosis of the terminal portion of the esophagus—I prefer this term to "cardiospasm"—the back pressure in the esophagus causes the musculature above the tubular narrowing to hypertrophy. This hypertrophy may be confined to the region just above the tubular portion or may extend any distance up the esophagus. It may involve both muscular layers or only the circular layer. When the tubular narrowing becomes extreme and especially when it is complicated by a backward bend of the terminal portion, the esophagus



Fig. 514—Multiple fibrous strictures beginning at the lower border of the cricoid cartilage. Such strictures have been found to be even more common than fibrous strictures at the lower end of the esophagus. The writer believes that the infection which causes them originates in the pharynx or the apices of the lungs.

septic pleurisy and pneumonia following perforation of the esophagus by a foreign body or by instrumentation.

I have summed up the question of cardiospasm for myself as follows:

The essential lesion is a tubular narrowing of the terminal portion of the esophagus in the crural canal and especially at the crural ring. This is due to a fibrosis of the periesophageal connective tissue and of the musculature of the esophagus and is caused by infection. The cause of the infection, if it originates within the esophagus, is probably ulcer. If it comes from without the thorax and the upper abdomen are the commonest sources. Infection picks out the terminal portion of the esophagus for attack because the anatomical configuration of this part makes it especially vulnerable. The fibrosed tubular portion seldom or never narrows enough to completely close the lumen of the esophagus. Complete closure is brought about by spasm or by a backward trap-like bend—I prefer this second explana-

tion. dilates progressively and sags to the right. Stagnant food produces ulcers of the mucous membrane, and the esophagus becomes chronically infected. The motor nerves—Auerbach's plexus—become infected and fibrosed. As a result the esophagus loses its muscular power. As it steadily dilates it becomes thinner and thinner and in the end is practically disorganized.

Spasm may initiate a certain number of cases of fibrosis of the terminal portion of the esophagus and may complicate any case at any time. In very few of the cases which I have treated was there proof that it played a major part.

Diagnosis—In fibrosis of the terminal portion of the esophagus the findings with the barium striped bag consist of a waist like narrowing of the terminal portion of the esophagus or two dents which are opposite each other and which give the impression of a stringlike stric-

ture The dent on the left is the larger and the more blunt It corresponds exactly to the shape and position of the edge of the left crus When a patient with cardiospasm drinks barium and then takes a forced inspiration there is a much

the crural crease was above the level of the crural opening, even above the level of the left half of the diaphragm The explanation of this high position of the crease I believe lies in the fact that the diaphragm has fallen, taking the

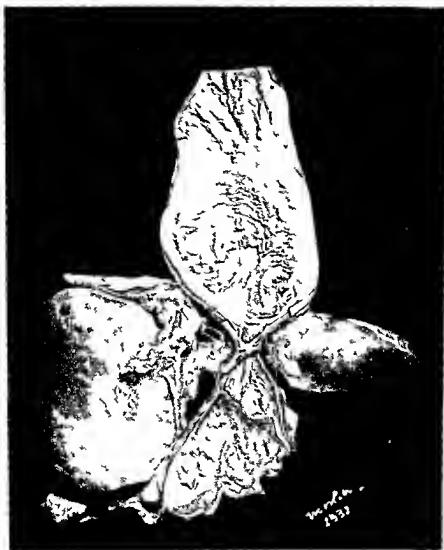


Fig 515—An extreme case of fibrosis of the terminal portion of the esophagus from infection from neighboring organ (front view) The patient from whom this specimen was obtained was treated by the writer in the early days of his work on the esophagus It was a typical case of what was then called cardiospasm There was a double bend at the terminal portion of the esophagus The fibrotic area was dilated with difficulty The patient refused further treatment The amount of dilatation obtained allowed the patient to live on soft solids At the end of ten years she died of suffocation from the overflow of liquid from her esophagus The specimen showed a small scarred liver, and a chronically infected gallbladder There was a large bandlike adhesion on the front of the terminal portion of the esophagus and multiple adhesions posteriorly The microscopic section of the constricted terminal portion of the esophagus showed that the musculature was almost completely replaced by fibrous tissue

larger impression on the left wall of the esophagus At first I considered this the impression of the left basal lung tip which is in the right position automatically to make such an impression, I now think it is the impression of the left crus A few times the bag has shown that

crural opening with it and leaving the fibrotic crease in the esophagus in its original position. (See Fig 519)

In order to make a gas bubble in the stomach so that I could get a clearer picture of the lower end of the esophagus in cases of fibrosis and

could see the bag more clearly, I began giving patients an effervescing Seidlitz power. This accomplished the purpose admirably. It also proved to be useful in another way. As a routine, I start my examination before the fluoroscope by giving the patient a few mouthfuls of

the barium is not forced through, so that the use of the powder also gives a rough idea of the tightness of the closure.

Treatment—The ideal treatment of narrowing of the terminal portion of the esophagus is dilatation under fluoroscopic vision. Mild

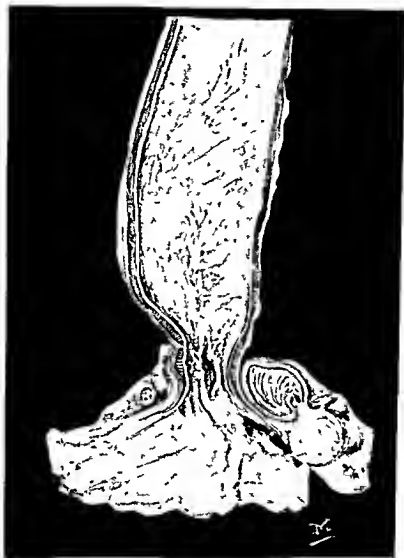


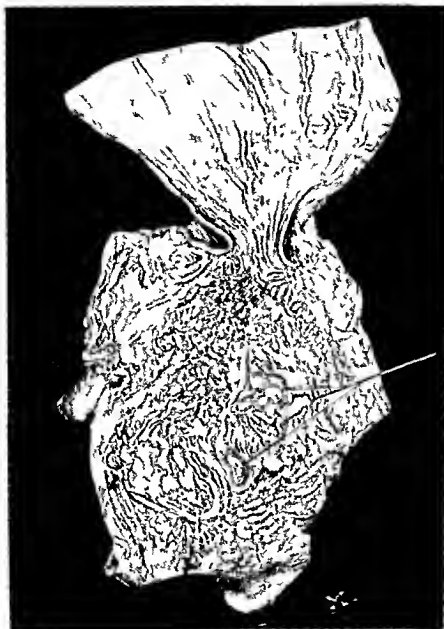
Fig. 516.—Autopsy specimen. The patient was a woman forty-five years of age. The specimen shows the typical narrowing of the terminal portion of the esophagus between the crura. Above this narrowing the esophagus is moderately dilated. The left crus is very large. On the right a septum completely divides the esophageal wall and the esophageal wall bends sharply to the right above it. The mucous membrane is intact. There are no ulcers. The crescentic black spot in the terminal portion of the esophagus is the traumatic perforation.

barium milk in order to find the fluid level in the esophagus. The barium settles to the constricted end of the esophagus. On giving the Seidlitz powder, however, the barium at once passes into the stomach, leaving the esophagus relatively clean for the passage of the bag. Occasionally the constriction is so tight that

cases of fibrosis respond readily. Even in cases of long standing, sufficient dilatation to give a clinical cure may be secured. In old cases in which the esophagus is much dilated and sags markedly to the right, dilatation—always under sight—may be tried cautiously. In such patients the esophagus is generally thin and its

musculature degenerated. These cases are dangerous to handle by dilatation. The patients are candidates for gastrostomy or for an anastomosis between the lower end of the

bag so that the shape which the bag assumes can be seen through the fluoroscope and dilatation can be carried out by sight. The lines are made with a mixture of barium powder and



Ulcers

Fig 517—Autopsy specimen. The patient was a man of seventy five years of age. The specimen shows the usual narrowing of the terminal portion of the esophagus. Above this the esophagus is much dilated. It is thin throughout, in places paper thin. The stomach is covered with thick white plaques. There are numerous superficial ulcers. Microscopical sections show that the muscular layers of the dilated esophagus are very thin everywhere infiltrated with small round cells and in places almost disintegrated.

esophagus and the fundus of the stomach (Figs 518, 519).

In dilatation under fluoroscopic vision the modified Sippy bag is recommended. The essential change in this bag from the original is the addition of four barium lines painted on the

rubber cement. On the end of the bag is a flexible finder, 4 or 5 inches long, with a heavy tip, the diameter of which is about $\frac{1}{4}$ inch. A finder of this size has only once failed to pass into the stomach, showing that the stricture in cases of cardiospasm is of large size. By intro

ducing the bag under the fluoroscope we can watch the finder turn "dead man's curve" and see when the end of it enters the stomach (The tip of the ordinary flexible bougie will not make

almost always occurs at the point just mentioned I have named this turn "dead man's curve") The present finder has a very heavy end which more or less automatically falls into

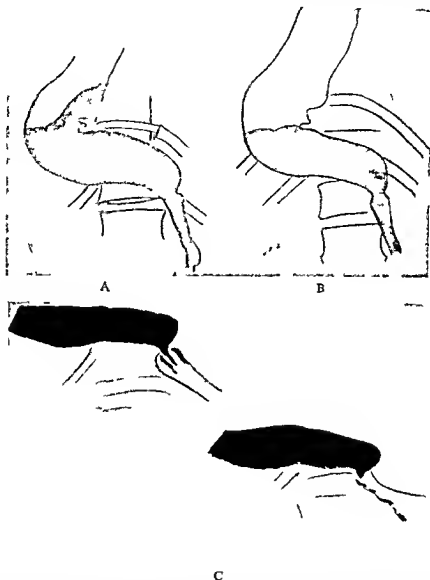


Fig 518 —Tracings of roentgen ray films of a long standing case of cardiospasm This patient has been treated for at least twenty years by periodically swallowing a string and having a perforated olive tipped bougie passed on it In A and B the esophagus is seen to be bent sharply to the right This is the typical formation in long standing cases of fibrosis The knee of this bend the writer calls "dead man's curve" because a bougie is so liable to scratch the esophagus at this point or to perforate it In B notice the ringlike narrowing at the terminal portion of the esophagus In C, the diaphragm is up There is an apparent invagination of the narrowed esophagus into the beginning of the stomach This patient was in one of the medical wards of the hospital for a heart and lung condition Passage of the barium bag was not attempted The writer believes that when the esophagus in a case of fibrosis reaches this stage of extreme deformity the patient is a candidate for an anastomosis of the lower end of the esophagus and the fundus of the stomach

the turn, hence it is the favorite site for perforations The blind passage of the old-fashioned elastic bougie has been accountable for many deaths, and, since the perforation

the stomach It is only when the finder is well in the stomach, of course, that it is safe to carry the bag down (See Figs 518-521)

The mercury bougie represents a great ad-



Fig 519—Reproduction of a print of a roentgen ray film. The patient is a man of sixty who has had difficulty in swallowing for thirty years. The esophagus is much dilated, bends sharply to the right, and the terminal 4 inches lie flat on the diaphragm. It is practically the same type of esophagus as that shown in Fig 518. In one of the barium roentgen ray films a horizontal ridge appeared in the lower part of the esophagus. This the writer considers a sign of previous ulcer. The film—right and left are reversed—shows a very marked crural crease. Dilatation with a pressure of 4 pounds gives this patient relief for about three weeks. He has declined to consider an operation for anastomosing the esophagus with the fundus of the stomach.



Fig 520—The Sippy dilator. The rubber bag of the dilator is held inflated, and its surface is quartered by four longitudinal lines painted on it with a mixture of equal parts of powdered barium and rubber cement. The lines are painted on the bag so that the dilatation can be carried out by sight. The lines will stay on the bag three or four weeks. After the application of the barium lines, the silk cover is drawn on. The bag is passed by touch until about a third the way down the esophagus. Then, under the guidance of the fluoroscope, the bag is so placed that the lower half is in the stomach and the upper half in the esophagus. The bulb from a blood pressure apparatus is used to dilate the bag with air, and the manometer from the same apparatus indicates the amount of air pressure in the bag. It has been found that a pressure of 3 or 4 pounds is sufficient in most cases to obliterate the constriction of the esophagus shown by the diagnostic bag. A pressure of 6 pounds gives severe pain, and the writer has used this amount but once. In one of his cases a pressure of $1\frac{1}{2}$ pounds was sufficient.

vance in the treatment of fibrosis of the terminal portion of the esophagus and of caustic strictures when they have been dilated sufficiently to

admit its passage. It is made in various sizes. In fibrosis of the terminal portion of the esophagus, after a certain amount of dilatation by

fluoroscopic sight has been obtained, the patient can be taught to pass the bougie himself. This saves many hospital visits, and much time both for the patient and the physician. In one of my patients the bougie opened and the mercury was discharged into the patient's stomach. The patient suffered no ill effects, however, he did not even have diarrhea.

In the future I shall take the liberty of adopting the principle of the mercury bougie in my modification of the Sippy bag. This mercury bougie I shall use to replace the whale bone staff, it will be a great improvement. Such a dilator was gotten out a few years ago and is much easier to pass than my model. However, it does not permit dilatation by sight, which I still consider the only thoroughly scientific way to carry out dilatation of the terminal portion of the esophagus. It represents a partial return to the dark days of esophageal manipulations. The instrument, however, is an efficient dilator.

The hollow bougie with the flexible metal staff is the most recent addition to my armamentarium and my feeling is that it will prove valuable, especially to those who are not doing many esophageal examinations. It occasionally happens, in beginning the treatment of a patient with fibrosis, that the finder will readily enter the stomach but the tip of the diagnostic bag will not. In these instances the hollow bougie with the flexible metal staff is especially useful. Suppose the esophagus is markedly bent to the right, in such a case, the flexible bougie fails to follow this bend. Without the flexible finder it is very dangerous to pass this curve. The flexible finder will take the curve readily and can be observed under the fluoroscope as it makes the turn. When the finder has been manipulated into the stomach the flexible metal staff is inserted. This straightens out the bend of the flexible bougie and affords a straight and safe passageway through the cardia. Bougies of any size can be fitted with the flexible finder so that after the fibrosis has been dilated sufficiently with the diagnostic bag, bougies of large size, equipped with the finder, can be passed instead of the bag.

I now use the large esophagoscope with the ballooning attachment only when there is a question of ulcer or cancer. In corrosive strictures, of course, I use the esophagoscope as of old.

In the treatment of fibrosis of the esophagus the first procedure is to cocaine the pharynx.

Then, after gaining the patient's confidence and exposing a preliminary roentgen ray film, the diagnostic bag is passed to locate the position of the fibrosis, to determine its amount, and to begin the dilatation. In the usual case three or four dilatations with a pressure of 3 or 4 pounds, at intervals of two or three weeks, will bring about sufficient dilatation of the fibrotic area to do away with most of the difficulty the patient has encountered in swallowing. When the esophagus can be dilated to normal, that is, up to 1 inch, under the pressure given above, the patient is instructed in the use of the mercury bougie and given one to use himself. In the beginning he generally passes the bougie once a day for a month or two and then gradually lengthens the interval. At first when the patient returns for observation and dilatation there is a fluid level at or near the arch of the aorta, showing that the backward bend plus the vertical twist keep up a certain amount of obstruction. A time comes, however, when the effect of the dilatation is permanent and the fluid line disappears. In other words, the patient comes in for examination with the esophagus empty. The majority of cases of fibrosis of the terminal portion of the esophagus can be successfully handled in this manner. The procedure, in a way, is somewhat complicated. It requires a sympathetic and cooperative roentgenologist and often much patience both on the part of the patient and the surgeon. Blind dilatation with a balloon bag is much easier and now is more often used. To my mind it is less scientific.

The method employed in a recent case (September, 1944) of Dr. Sweet's of the Thoracic Service of the Massachusetts General Hospital may revolutionize the treatment in the ordinary case of fibrosis of the terminal portion of the esophagus and the patient may be spared prolonged dilatation. The case referred to was one of Dr. Harold Tohey's. It could be called a usual case of fibrosis of the terminal portion of the esophagus. Treatment by dilatation had not been tried. Dr. Sweet excised the fibrosed and narrowed portion of the esophagus and made an anastomosis between the esophagus and the stomach.

In old cases of fibrosis in which the esophagus is much dilated and in those in which the terminal portion of the esophagus has a double twist, I have for years advised an anastomosis between the esophagus and the stomach. I succeeded in dilating one such esophagus but the

procedure was long and was attended by much anxiety

When fibrosis of the terminal portion of the esophagus has existed for as many as thirty years (I have such a patient), the esophagus is markedly dilated and sags decidedly to the right. The terminal portion bends to the left and lies horizontally on a flat motionless diaphragm. Just before the esophagus gains the

that it is better, at least for a novice, to have the patient swallow a string and to use this as a guide in passing the bag. Furthermore, in these cases, unless the bag can be manipulated into the stomach either by sight or by the help of a swallowed string, I feel that the operation of anastomosing the terminal portion of the esophagus to the fundus of the stomach is the coming method of treatment. A number of such oper-

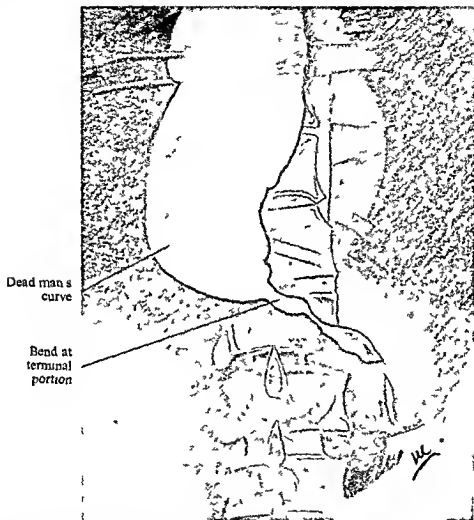


Fig. 521—Retouched tracing from roentgen ray film, demonstrating the typical picture in an advanced case of fibrosis of the terminal portion of the esophagus. A no. 35 French elastic bougie would pass easily. The patient was a man, fifty five years of age who had had fibrosis of the esophagus for fifteen years. This patient died from tuberculosis of the lungs. (See Fig. 518.)

stomach it bends in gooseneck fashion back to the right. In other words, the esophagus has two bends. These cases are dangerous to handle with the bag unless extreme care is used. Often it requires great patience to coax the spiral wire finder into the stomach, and in some of the cases the fibrotic opening is so narrow that it will admit only the tip of the bag. In these old cases the esophageal wall is extremely thin, so

ations have been done in England and at least two in the Massachusetts General Hospital by Dr. Churchill. The difficulty in all work on the lower end of the esophagus by the abdominal route, up to the present time, has been the inadequate and restricted surgical approach. However, by turning up the left lobe of the liver and mobilizing the lower end of the esophagus Dr. Churchill demonstrated that the operative

field can be much enlarged, and in the case in which I saw him use this procedure he had ample room to work. This is a great advance in operative technic.

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ESOPHAGOLOGY

The esophagus, from the anatomic and autopsy viewpoints, was well known to Hippocrates and Galen, and for twenty centuries it was studied by many great physicians and surgeons, chiefly from the same viewpoints, but clinically, the esophagus remained a discouraging problem. Near the end of the nineteenth century the esophagoscope was devised, but the mortality from its use made clinical studies on the living of slow progress. Early in the twentieth

century the instrumental equipment and the technic of esophagoscopy were improved, necessity for training to avoid the pitfalls was recognized, the American Bronchoscopic Society, now called the American Broncho Esophagological Association, was formed—all these factors enormously increased interest in and knowledge of the clinical phases of esophageal diseases. The studies of Mosher and other members of the society mentioned have placed our knowledge of these diseases on a sound pathologic basis. The studies and cooperation of the roentgenologists have added greatly to our knowledge of the physiology and diseases of the esophagus. Altogether the sum of knowledge has reached the point at which *esophagology* is recognized as a Department of Medical Science.

ESOPHAGOSCOPY

Esophagoscopy means "examination of the esophagus with an endoscopic tube." This examination necessarily includes the hypopharynx and should include the examination of the folds of the stomach in the region of the esophageal entrance. In principle it is a specular examination, in practice more training is necessary for safe and satisfactory use than is required for most other specula.

Indications—Esophagoscopy examination is indicated in every patient who complains of the slightest abnormality in the swallowing function or abnormal sensation in the region of the esophagus, the hypopharynx, or the larynx. It is often indicated as a preliminary to the first blind passage of a flexible lens-system gastro-scope.

Contraindications—There are no absolute contraindications if esophagoscopy is urgently indicated, in a case of foreign body for example. There are, however, a number of contraindications that might outweigh less urgent indications. First and most important of these is lack of a physician who has learned how to avoid the dangers of untaught esophagoscopy. When a trained esophagoscopist is available an esophagoscopy examination may be requested with no more hesitation than any other specular examination. One condition that renders esophagoscopy inadvisable, except for foreign body, is aneurysmal esophageal compression as revealed by a fluoroscopic examination. Skillfully done, there is no danger of rupturing an aneurysm unless rupture is imminent, but fluoros-

scopic diagnosis of aneurysmal compression stenosis is a conclusive diagnosis and one ample for planning all the treatment medical science can offer the patient. This, however, does not apply to any other form of esophageal obstruction. Conditions that call for postponement of esophagoscopy until after a course of preparatory treatment are dehydration, acidosis, partial pneumothorax on one side with complete pneumothorax on the other, subcutaneous or mediastinal emphysema, perforation of the hypopharyngeal or esophageal wall from instrumentation, a high degree of hypertension. Unless due to tracheal compression by a large

larynx and eyes and over the operator and the floor.

Anesthesia—Esophagoscopy is not painful; therefore anesthesia is not necessary and should not be used for routine repetitions of treatment such as is required by cicatricial stenosis. For example, for a first diagnostic examination the pharynx may be locally anesthetized by dropping into it 2 cc (32 minims) of a 10 per cent watery solution of larocaine, repeated three times at intervals of three minutes. Four drops of a 4 per cent cocaine solution may be used in the same way. When the dropper method is used the patient should be recumbent. Larocaine

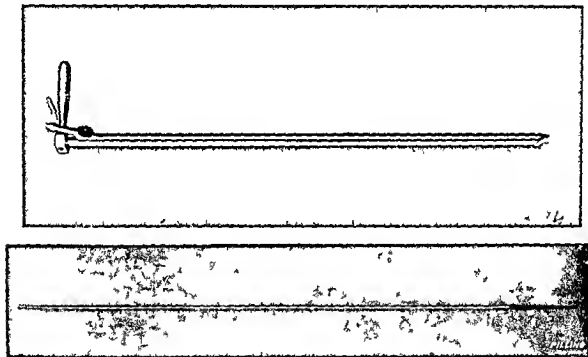


Fig 522.—The esophagoscope here shown is made with integral drainage canal for keeping field clear of secretions without interruption during esophagoscopy. Below is shown a silk woven bougie for esophagoscopic bougienage.

foreign body in the esophagus, severe dyspnea should be taken care of before esophagoscopy, and even in such a case proper provision should be made for increase of dyspnea (q.v.).

Preparation of the Patient for Esophagoscopy—An empty stomach and the sermon on relaxation (q.v.) are the essentials. In case of dilated esophagus with a chronic tendency to accumulate food the esophagus should be emptied by lavage every evening for three or four days and again by stomach tube an hour or two beforehand. Neglect of these precautions will result in strangling panic impending asphyxia and a mess of sour food in the patient's

crine being nontoxic the patient may be given a larocaine lozenge to dissolve in his mouth. Premedication with morphine sulfate $\frac{1}{4}$ grain (0.02 gm.) given hypodermically an hour beforehand helps the apprehensive adult patient to relax at a first esophagoscopic examination. Practically all patients prefer to dispense with both local anesthesia and premedication after the first few esophagoscopies. Lack of rigidity in children lessens the need for anesthesia. A proportionate dose of premedication may be used at the first few esophagoscopies if desired. If an inexperienced operator is compelled by force of circumstances to esophagoscope a

muscular adult with a large foreign body in the esophagus the patient will be in less danger if fully relaxed by deep ether anesthesia. Sodium amylal and avertin are unsuitable because

sary because there is no pain associated with esophagoscopy.

Instruments for Esophagoscopy.—A standard type of esophagoscope is shown in Figure 522

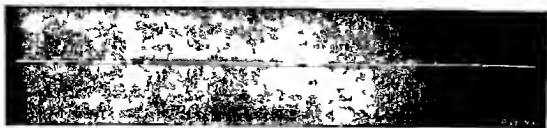


Fig 523 —Lumen finder for finding the lumen ahead at esophagoscopy. It is not a mandrin, and is never used as such. It is used only under guidance of the esophagoscopist's eye at the proximal tube-mouth.

they increase muscular contractions. It is only relaxation that is needed. Analgesia is unneces-

The sizes required for adults and children and the various accessory instruments are listed along with the equipment for bronchoscopy. One special instrument of great importance for safe passage of the esophagoscopy is the lumen finder shown in Figure 523. Forceps for esophagoscopy are the same as those illustrated for bronchoscopy in Figure 452 (p. 610).

INSINUATION OF THE ESOPHAGOSCOPE

Seven fundamental rules are essential for safety, and must be thoroughly memorized before attempting to make an esophagoscopic examination.

1 An esophagoscope is *insinuated* not merely 'introduced'. If put into the pharynx and pushed upon, the one place into which it will not go is into the esophagus.

2 A lumen must always be located ahead before the tube is advanced; if not visible it must be found with the lumen finder.

3 The first and *greatest danger point* is where the esophagus is pinched shut by the cricopharyngeus muscle (Fig. 524).

4 The *second danger point* is where the esophagus is pinched shut at the hiatus oesophageus by the diaphragmatic musculature pulling upon the crura. These two points have been called *pinchcocks* because their pinching shut of the esophageal tube from the outside is similar in mechanism to that of the pinchcock used in laboratories to pinch shut the rubber tube on the buret. In over 10,000 esophagoscopies without anesthesia the writer has never seen a total absence of this pinching at these two points unless there was paralysis or local disease.

5 At both these points the esophagus is tonically closed at all times except when a bolus of food or a gulp of liquid approaches and makes a reflex demand for opening. They are automatic gates that normally open to permit passage of each unit of traffic and promptly close after each unit has passed.

6 The esophagoscope must await the opening of these tonically closed points while it is continuously but gently pressed in exactly the right place. The gates must

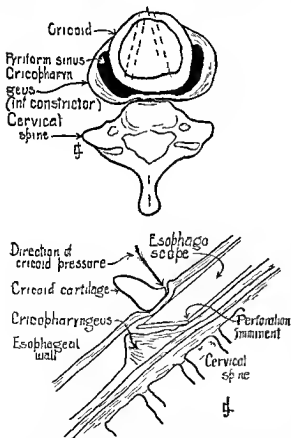


Fig. 524—Schematic illustration of the anatomic basis for difficulty in introduction of the esophagoscope. The cricoid cartilage is pulled backward against the cervical spine, by the cricopharyngeus, so strongly that it is difficult to realize that the cricopharyngeus is not inserted into the vertebral peristome instead of into the median raphe. This external musculature pinches the esophagus shut like the pinchcock on the rubber tube of a buret, and it has therefore been called the cricopharyngeal pinchcock.

not be crashed For him who tries to crash it the cricopharyngeal gate is a Gate of Tears (Bab el Mandeb)

7 The lumen finder (Fig 523) will locate the spot and the passage of the finder will usually cause the respective musculatures to relax and open Size 10F is best for this

The following is a description of the standard technic of esophagoscopy as used at the Chevalier Jackson Clinics for many years¹

Position of the Patient—The patient's vertex must be about 15 cm above the level of the table for the esophagoscope to pass through the cervical esophagus, and 2 or more centimeters below the level of the table to pass through the abdominal esophagus, because of the plane of the axes at the respective locations the patient's

the anterior wall The larynx is integral with the anterior hypopharyngeal wall, but is held closely against the posterior hypopharyngeal wall by the action of the inferior constrictor of the pharynx, and particularly by its specialized portion—the cricopharyngeus muscle (Fig 524) A bolus of food is split by the epiglottis and the two portions drifted laterally into the pyriform sinuses, the recesses seen on either side of the larynx But little of the food bolus passes posterior to the larynx during the act of swallowing It is through the pyriform sinus that the esophagoscope is to be inserted, thereby following the natural food passage To insert the esophagoscope in the midline, posterior to the arytenoids, requires a degree of force dangerous to exert and almost certain to pro-

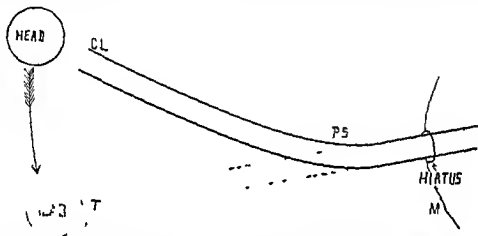


Fig 525—Schematic illustration of the high low method of esophagoscopy fourth stage Passing the hiatus The head is dropped from the position of the first and second stages CL to the position T, and at the same time, the head and shoulders are moved to the right (without rotation) which gives the necessary direction for passing the hiatus

head must be high and then low (Fig 525) Thus "high low sequence" requires that the patient's head and neck be out in the air away from the table and supported by an assistant, who also holds the bite block (Fig 343) A second assistant holds the shoulders of the patient down on the table These details are the same as in bronchoscopy (q v) On the proper work of these two assistants depends the smoothness of the technic of introduction Their work is not merely the restraint of the patient, it is the placing of the passages in proper line for introduction of the tube They must understand this purpose and how to accomplish it

Entrance to the Esophagus.—The hypopharynx tapers down to the gullet like a funnel, and the larynx is suspended in its lumen from

duce damage to the crico arytenoid joint or to the pharyngeal wall or to both

Handling of the Esophagoscope—The esophagoscope is steadied by the left hand like a billiard cue, the terminal phalanges of the left middle and ring fingers hooked over the upper teeth, while the left index finger and thumb encircle the tube and retract the upper lip to prevent its being pinched between the tube and upper teeth The right hand holds the tube in pen fashion at the collar of the handle, not by the handle During introduction the handle is to be pointed upward toward the zenith This is not only for safety (Fig 524) but to keep the drainage canal at the bottom of the field, a dry field is invaluable for both success and safety No lubrication is used on the esophagoscope, the secretions are sufficient

Technic—For clearness of description the technic is divided into four steps (1) entering the right pyriform sinus, (2) passing the cricopharyngeal gateway, (3) passing through the thoracic esophagus, and (4) passing through the hiatal gateway. The endoesophageal appearances at these points are shown in Figure 346.

Step 1 Entering the Right Pyriform Sinus The operator standing (Fig. 526) inserts the esophagoscope along the right side of the tongue as far as and then

Step 2 Passing the Cricopharyngeal Constriction This is the most difficult and dangerous part of esophagoscopy especially if the patient is unanesthetized. Local anesthesia helps little if at all. The handle of the esophagoscope is still pointing upward and consequently we are sure that the tip of the esophagoscope is directed anteriorly. Force must not be used, but steady moderately firm pressure against the tonically contracted cricopharynx is made while at the same time the distal end of the esophagoscope is lifted anteriorly by the left thumb. After a few seconds sometimes a minute of waiting a lumen will usually appear in the



Fig. 526—Esophagoscopy by the high low method first step. High position. Finding the right pyriform sinus. In this and the second stage the patient's vertex is about 15 cm. above the level of the table. Note the operator's left hand with fingertips in the patient's mouth; the thumb is posterior to the tube ready to lift the tube forward. The battery table is shown in its correct relation to the operator.

down along the posterior pharyngeal wall. A lifting motion imparted to the tip of the esophagoscope by the left thumb will bring the rounded right arytenoid eminence into view. This is the landmark of the pyriform sinus and care must be taken to avoid possible injury that might occur from hooking the tube mouth over it or its fellow. The tip of the tube should now be directed somewhat toward the midline remembering the funnel shape of the hypopharynx. It will then be found to glide readily through the right pyriform sinus for 2 or 3 cm when it comes to a full stop and the lumen disappears. This is the tonically closed cricopharyngeal constriction—the cricopharyngeal gate.

upper portion of the endoscopic field. To make sure of a lumen ahead a lumen finder (Fig. 523) is inserted; a small size is best; it permits the operator to see past it. This not only makes sure of the lumen but it causes the cricopharynx to relax and thus permits the wide opening of the muscular constriction. The general direction of the axis of the entire esophagoscope must be maintained as if aiming for the midline of the thorax as indicated by the midline of the manubrium (but posterior thereto) notwithstanding the start is from the right side, i.e. from the right pyriform sinus. Pointing over to the left is one of the chief errors of direction and with undue pressure it may contribute to the making of

a perforation. At this stage great care is necessary on the part of the assistants to be sure the head is high and the shoulders of the patient do not rise from the table, the 'sermon on relaxation' should be repeated quietly in the patient's ear by the assistant holding the head. The tip of the esophagoscope enters the opening lumen and the slanted end slides over the fold of the cricopharynx into the cervical esophagus. There is usually from 1 to 3 cm. of this constricted lumen at the level of the cricopharynx and the subjacent orbicular esophageal fibers.

Step 3. Passing through the Thoracic Esophagus The lumen of the thoracic esophagus will be seen to enlarge during inspiration and diminish during expiration due to the change in intrathoracic pressure when air is

top (Fig. 528). Flexion of the legs at this stage may help to relax the hiatal constriction. The tube will now be pointing in the direction of the anterior superior spine of the left ilium. To find and pass through the hiatal gateway requires a clear conception of the physics of this part of the gullet in order to understand the endoscopic images and the difficulties encountered. It should be recalled that the diaphragm is a floor through which the esophagus we are exploring penetrates at a certain place called the hiatus oesophageus and further that this hiatus does not gape. It is pinched shut by the hiatal pinchcock which is a mechanism external to the esophageal wall just as the pinchcock of the buret is external to the rubber tube. As seen endoscopically the hiatal constriction may assume the form of a slit or rosette or



Fig. 527—High low technic third step passing through the thoracic esophagus. The operator follows the lumen and the assistant at the head follows the operator. After the level of the heart is passed the esophagus turns forward and the head must be lowered to keep the lumen axially presented before the tube mouth.

admitted through the esophagoscope. The esophagoscope usually glides easily through the thoracic esophagus if the patient's position is correct. After the levels of the aorta and left bronchus are passed the lumen of the esophagus seems to have a tendency to disappear anteriorly. The lumen must be kept in axial view and the head lowered as required for this purpose (Figs. 525, 527).

Step 4. Passing through the Hiatal Gateway When the head is dropped it must at the same time be moved horizontally to the right in order that the axis of the tube shall correspond to the axis of the lower third of the esophagus, which deviates to the left and turns anteriorly. The head and shoulders at this time will be found to be considerably below the plane of the table

of one two three or more folds. If the rosette, slit, or folds cannot be promptly found as may be the case in various degrees of diffuse dilatation the tube mouth is shifted usually farther to the left and anteriorly, the lumen finder will then safely locate the hiatus. When the tube mouth is centered over the hiatal constriction, moderately firm pressure continued for a short time will cause it to yield. Then the tube maintaining this same direction will without further trouble pass into and through the abdominal esophagus. The cardia will not be noticed as a constriction but its appearance will be announced by the rolling in of reddish gastric mucosal folds and by a gush of fluid from the stomach. Retching is not usually troublesome while the gastroscope is in the stomach; it is more likely to occur while the tube

mouth is seeking the hiatus above the diaphragm and cough may be noted at the same time (The foregoing description of 'Step 4' is typical of esophagoscopy without anesthesia.)

Normal esophageal mucosa under proper illumination is glistening and of a yellowish or bluish pink. The folds are soft and pliable, rendering infiltration quickly noticeable. The cricoid cartilage shows pallidly through the mucosa. It must be remembered that the mucosa varies in color within the normal limits. In anemic individuals it is a whitish pink, the

toward crimson. As compared to the esophagus, vessels are less visible in the gastric mucosa, but they can usually be seen. In both the esophagus and stomach mucosal vessels may be rendered invisible by inflammatory conditions.

Secretions in the esophagus are readily aspirated through the drainage canal by a negative pressure pump. Food particles are best removed by "sponge pumping" or with forceps. Should the drainage canal become obstructed positive pressure from the pump will clear the canal. The handle of the esophagoscope should be up at



Fig. 528.—Esophagoscopy by our high low method. fourth stage. Passing the hiatus. The patient's vertex is about 5 cm. below the top of the table. The esophagoscope is pointing toward the anterior superior spine of the patient's left ilium. The patient's knees may be flexed if desired to assist relaxation.

engorged mucosa of the plethoric patient may be so red as to be mistaken for a diseased condition. The color of each type will vary with the degree of illumination. As in any other kind of direct examination nothing will take the place of education of the eye to the normal, and especially to the variations within normal limits. Vessels are visible in the normal mucosa running chiefly in a longitudinal direction and branching freely. The gastric mucosa is a darker pink than that of the esophagus and when actively secreting, its color in some cases tends

all times to keep a dry field. If turned down for temporary use of the lip of the tube mouth it should be promptly restored to the up position. This brings the intake orifice at the distal end lowermost in standard tubes, when using full-lumen tubes the handle should be brought to point toward ten o'clock to lower the intake orifice.

Dyspnea during Esophagoscopy.—In case of a child dyspnea during esophagoscopy may be due to the use of too large a tube, which encroaches upon the lumen of the trachea by

pressing forward the "party wall." The same encroachment, in either adults or children, may be due to faulty position of the patient (head too low), or instrument, or to overriding the foreign body. It may also be due partly to an enlarged thymic gland anteriorly as shown by Ellen J. Patterson. If no general anesthetic has been given, and the tracheal occlusion is not kept up too long, no danger is involved, but under general anesthesia a death on the table is imminent. An enlarged thymus gland enormously increases the risks of dyspnea during esophagoscopy. It must never be forgotten that the trachea of a baby is compressed to obliteration of the lumen as easily as the rubber of a medicine dropper.

Difficulties.—The two greatest difficulties in passing an esophagoscope when the patient has not had a general anesthetic are at the two points where the esophagus is normally pinched shut by the normal physiological musculature that keeps these two points always tonically closed. Anyone who does an esophagoscopy in a patient who is under general anesthesia and then goes to the cadaver to count the muscular fibers at these points gets no conception of the tight clamping shut of these two physiologic gates in the pathway of the esophagoscope. They are often indicated in the fluoroscope as a momentary hesitation of opaque food in swallowing, but the normal reflex arc is usually so perfect in timing and sequence that the gate is open by the time the opaque bolus reaches the gateway. Naturally the gate does not open so spontaneously at the approach of the metallic, rigid esophagoscope passed with all the entire muscular system tensed by the procedure. The difficulty at both the gates is overcome by gentle insinuation of the lumen finder. This does two things—it brings the distal end of the esophagoscope into proper position before the gate, and its passage induces the gate to open. If continuous but gentle advancing pressure is made on the esophagoscope when the lumen finder locates the gateway the esophagoscope will slip through promptly, if there is no organic stenosis. All of the difficulties at these two physiologic gateways are eliminated by complete muscular relaxation of deep anesthesia of inhaled ether, and it would be well for the inexperienced to use this kind of anesthesia for adult patients, in his first few dozen esophagoscopies. It would be fatal from asphyxia in a child with a large foreign body in the cervical

esophagus for reasons given in a previous paragraph. There is no danger of this in an adult. An emaciated, edentulous, elderly patient would be best for a first case without anesthesia. After having acquired experience, and with the patient held in proper position by assistants who also have had experience, the esophagoscopist can insinuate the esophagoscope all the way through into the stomach of any patient whose mouth can be opened and whose esophagus is not obstructed by an organic lesion.

Apart from the two greatest difficulties mentioned in the foregoing paragraph the inexperienced esophagoscopist may find one other. The esophagoscope may seem to be fixed so rigidly that it can be neither introduced nor withdrawn. This usually results from a wedging of the tube in the dental angle, it occurs oftenest and is most troublesome in patients who have narrow upper and lower alveolar arches. It can be overcome by a wider opening of the jaws, or perhaps by easing up of the bite block, but most often by correcting the position of the patient's head. A smaller esophagoscope may be used. Here again relaxation under deep general anesthesia will help, and should be used when the inexperienced operator notes narrow alveolar arches at the preliminary throat examination. Parenthetically it should be stated that some of the much used narcotics do not produce complete muscular relaxation.

A few minor matters may be mentioned. In cases of kyphosis it is a mistake to try to straighten the spine. The head should be held correspondingly higher at the beginning, and should be very slowly and cautiously lowered. Once inserted, the esophagoscope should not be removed until the completion of the procedure, unless respiratory arrest demands it. Occasionally, in stenotic conditions, the light may become covered by the upwelling of a flood of fluid, and it will be thought that the light has gone out. As soon as the fluid has been aspirated the light will be found burning as brightly as before. If a lamp should fail it is unnecessary to remove the tube, as the light carrier and lamp can be withdrawn and quickly replaced by the duplicate carrier and lamp, always kept sterile and ready on the instrument table. A complete instrumental equipment with proper selection of instruments for the particular case is necessary for smooth working. Such an equipment is listed under the head of "Bronchoscopy." In a busy clinic, duplicates of

the frequently used instruments save much time that would be lost in cleaning and sterilizing.

Introduction of the Esophagoscope through the Laryngoscope—The esophagoscope may be introduced through the laryngoscope in the same way as the bronchoscope is introduced. The right pyriform sinus is exposed by the same method as in bronchoscopy (Fig 457) then the esophagoscope is introduced into the right pyriform sinus and past the *cricopharyngeal* fold. The laryngoscope may then be separated and removed. Thence downward the esophagus is explored as in the method previously described.

Specular Esophagoscopy—Inspection of the hypopharynx and upper esophagus is readily made with the esophageal speculum. High lesions and foreign bodies lodged behind the larynx are thus discovered with ease and such a condition as a retropharyngeal abscess which has burrowed downward is much less likely to be overlooked than with the esophagoscope. High strictures of the esophagus may be exposed and treated by direct visual bougienage until the lumen is sufficiently dilated to allow the passage of the esophagoscope for bougienage of the deeper strictures. One drawback to the use of specula is the same as to the use of short tubes namely if a foreign body should escape downward on specular examination it cannot be followed quickly enough to catch it before it shall have entered the stomach. This applies only to cases in which there is no stenosis.

Technic of Specular Esophagoscopy—The patient is placed in the recumbent position as shown in Figure 343. The larynx is to be exposed as in direct laryngoscopy. The right pyriform sinus identified and the tip of the speculum inserted therein and gently insinuated to the crico-pharyngeal constriction. Too great extension of the head is to be avoided—even slight flexion at the occiput to avoid joint may be found useful at times. Moderate anterior or upward traction pulls the cricoid away from the posterior pharyngeal wall and the lumen of the esophagus opens above a crescentic fold, the *cricopharyngeal* crescent. The speculum readily slides over this fold and enters the cervical esophagus. In searching for foreign bodies in the esophagus the speculum has the disadvantage of limiting length so that should the foreign body move downward it could not be followed. In case of suspected disease if no lesion is encountered high the speculum must be withdrawn and an esophagoscope introduced making two esophagoscopies instead of one. A negative opinion could not justly be based on the specular high examination alone.

Complications following Esophagoscopy—These are to be avoided in large measure by the

exercise of gentleness care and the skill that is acquired by practice. If the instructions herein given are followed esophagoscopy is practically without mortality apart from the conditions for which it is done. Injury to the crico-arytenoid joint may simulate recurrent paralysis. Posticous paralysis may occur from recurrent or vagal pressure of a misdirected esophagoscope. These conditions may persist but recovery usually takes place. Perforation of the esophageal wall may cause death from septic mediastinitis. Expert roentgen ray work will locate a purulent collection usually evacuation through the neck is feasible and relatively safe. The approach is the same as for pharyngeal diverticulum (*qv*) except that esophageoscopic aid is not used. Pain—in the back, back of the head and behind the sternum—with moderate fever are the symptoms. After recovery pain vague and scattered may be occasionally felt for years. The pleura may be entered—pyo-pneumothorax will result and demands immediate thoracotomy and usually also gas-troscopy though sometimes a nasal feeding tube will tide the patient over until the perforation heals. Aneurysm of the aorta may be ruptured. Patients with tuberculosis compensating cardiovascular lesions or other advanced organic disease may have serious complications precipitated by esophagoscopy.

Retrograde Esophagoscopy—The best position is dorsal with the patient flat on the table carefully avoiding the opisthotonos position and crural tension. Flexion of the knees may help by relaxing abdominal and diaphragmatic tension. A scaphoid abdomen is a great help. Empty intestines facilitate palpation of the spine for localization of the crura and hiatus as well as favor a better angle of approach (Fig 529). The first step is to get rid of the gastric secretions. There is always fluid in the stomach and this keeps pouring out of the tube in a steady stream. Fold after fold is emptied of fluid. Once the stomach is empty the search begins for the cardiac opening. The best landmark is a mark with a dermal pencil on the skin at a point corresponding to the level of the hiatus oesophageus. At retrograde esophagoscopy there seems to be no abdominal esophagus and cardia. The esophagoscope encounters only the diaphragmatic pinching shut which seems to be at the top of the stomach like the closing drawstring at the top of a bag. The tube mouth is moved over the stomach wall lying in close contact

with the vault of the diaphragm until the hiatus is felt through the stomach wall with the tube mouth, as a yielding depression. The lumen finder is as useful here as it is in approaching the hiatus from above. If the esophagus is not impervious a swallowed string may be brought out through the fistula; the retrograde esophagogastroscope is passed over the string and under ocular control goes directly to the hiatus. When it is desired to do a retrograde esophagoscopy and the gastrotomy is done for this special purpose, it is wise to have the incision near the median line and not very high. Once the hiatus

thelial sealing over the upper entrance of the stricture. In such cases the smallest size of our filiform bougies (Fig. 522) is inserted through the retrograde esophagoscope and insinuated upward through the stricture. When the tip reaches the pharynx coughing, choking, and gagging are noticed. The filiform end is brought out the mouth sufficiently far to attach a silk braided cord which is then pulled down and out of the gastrotomic opening. The no. 14 braided silk "string" must be long enough so that the oral and the abdominal ends can be tied together to make it "endless"; but before doing so



Fig. 529.—Retrograde esophagoscopy The esophagoscope has been passed into the thoracic esophagus. As it is advanced farther upward toward the cricopharyngeal pinchcock, lowering of the patient's head is necessary. Duodenoscopy requires only the turning of the esophagoscope and advancing it across the spinal hump on the posterior wall of the stomach to and through the pylorus. Gastrosocopy is a simple procedure. Using the same esophagoscope the entire interior of the stomach can be explored. Pyloroscopy, gastrosocopy, and duodenoscopy are routine procedures in cases in which gastrotomy has been necessary for esophageal stenosis.

is located and the esophagus entered, the remainder of the work is very easy. Bougienage can be carried out from below the same as from above and may be of advantage in some cases. Stricture lumina are much more likely to be concentric as approached from below because there has been no distortion by dilatation due to pressure of the stagnant food operating through a long period of time.

Retrograde esophagoscopy is sometimes useful for "stringing" the esophagus in cases in which the patient is unable to swallow a string because he is too young or because of an epi-

the oral end should be drawn through the nose where it will be less annoying than in the mouth. The purpose of the "string" is to pull up the retrograde bougies (Fig. 531).

In cases of total atresia perforation through from the peroral to the retrograde tube mouth may be accomplished with the expert assistance of a roentgenologist with a double-plane fluoroscope. The single-plane fluoroscope will prove disastrously misleading for this work. (See under "Biplane Fluoroscopy as an Aid in Bronchoscopy, Esophagoscopy, and Gastrosocopy.")

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TRAUMA OF THE ESOPHAGUS

Trauma of the cervical esophagus by penetrating and perforating missiles, and by stabs and gashes, has been considered along with trauma of the larynx. Transthoracic trauma of the thoracic esophagus is a problem of external thoracic surgery. Endoesophageal trauma occasionally occurs from bones swallowed in hasty eating or during intoxication. Children occasionally swallow rough or sharp toys accidentally. Manic depressive mental patients do so wilfully. Usually these wounds are superficial, though occasionally perforating trauma results, as elsewhere herein mentioned. We have seen cases of rupture of the esophagus from force of compressed air from a hose, also of water from a hose, rupture of the pulmonary alveoli accompanied the esophageal rupture in these cases, which all terminated fatally.

Instrumental trauma was common in pre-esophagoscopy days from the blind passage of bougies for diagnosis and of probangs used in efforts to push down or pull up foreign bodies. Usually instrumental perforation occurs from a failure on the part of the operator to realize that "the esophagus is surgically one of the most intolerant viscera in the body."¹ This peculiar intolerance begins at the cricopharyngeal level. It is difficult for the surgeon who operates freely in the air passages above and below this level, and extensively above this level in the food passages, to realize that as soon as he crosses this barrier he enters a region where trauma involves vital risks out of all proportion

to the extent of the trauma.¹ The slightest injury involving both mucosa and muscular coat is usually disastrous. This is not altogether due to the fact that the esophagus is a septic canal. It is no more so than the intestinal canal and its walls are no thinner, yet the work of the abdominal surgeon has no parallel in esophageal surgery. Another misconception that leads to disaster is that a morbid state, such as a stricture, though it may be strong, may not involve the whole periphery. An operator with a conception of a concentric cicatricial lumen over-stretches the normal wall section of a one-sided stricture, and a little separation of the fibers of the muscular coat with a tiny split in the mucosa is usually fatal. On the other hand, with knowledge gained by visual examination with the esophagoscope and slow, patient dilatation extending over many months, that small section of normal wall can be dilated to a full-sized lumen. Moreover this kind of dilated lumen will be permanent without any of the tendency of dilated cicatrices to recurrent contraction.

Rupture or perforation of the wall of the thoracic esophagus produces profound shock, fever, mediastinal emphysema, and rapid sinking. Pneumothorax and empyema follow perforation into the pleural cavity, saliva and food pass in through the perforation. Rupture of the cervical esophagus is usually followed by cervical emphysema and cervical abscess, both of which often burrow into the mediastinum along the fascial layers of the neck. Rupture of the thoracic esophagus may cause abscess that will extend upward into the neck. Lesser degrees of trauma produce esophagitis usually accompanied by fever, dysphagia, and odynophagia. Usually there is a momentary pain as any swallowed substance passes the traumatized location.

The treatment of traumatic esophagitis consists of rest in bed, sterile liquid food, and the administration of bismuth subnitrate, about 15 grains (1 gm.) in an adult, dry on the tongue every half-hour for six hours, then every three waking hours for a few days. Rupture of the esophagus requires immediate gastrotomy to put the esophagus at rest and supply necessary alimentation. Thoracotomy for drainage is required when the pleural cavity has been involved, not only for pleural exudates but for the constant and copious esophageal leakage. It is not ordinarily realized how much normal salivary drainage passes down the esophagus

One-way valve action, by the forcing of air through an esophageal perforation into the pleura, may cause a dangerous degree of positive air pressure in the thorax requiring release. A syringe needle might be used for this, but infection usually requires opening the pleural cavity. Mediastinal abscess may require evacuation. If the pleural cavity is uninvaded this is done, preferably, through the neck, above the clavicle. The surgical approach is the same as in excision of pharyngeal diverticulum (*qv*). The usual treatment of shock is required. No attempt should be made to remove a foreign body, if one is present, until the traumatic lesions have healed. This may require a number of weeks. Decision as to when to remove the intruder is determined by esophagoscopy inspection and roentgen ray study. Subcutaneous emphysema does not require puncture unless gaseous, or unless pus forms. In the latter event free external drainage becomes imperative. Chemotherapy (*qv*) may be indicated to control sepsis. In case of false passage from instrumental perforation, esophagoscopy is contraindicated until the false passage closes, if the patient should survive long enough for closure. Continuous wearing of a feeding tube is advisable in most cases to provide drainage of saliva and means of prevention of dehydration and starvation. The prognosis is always serious, but perhaps as many as a third of the patients survive under proper care.

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BURNS OF THE ESOPHAGUS

Incidence and Etiology.—Burns from swallowing hot liquids are rare. Chemical caustics, such as storage battery solutions, are sometimes swallowed by children or intoxicated persons. Burns from household lye, household cleansers, and drainpipe cleaners were frequent until the passage of the "Federal Caustic Act,"

compelling the use of conspicuous "Poison" labels, was accomplished by the years of work of the Committee on Lye Legislation of the Section on Laryngology of the American Medical Association.^{1 2 3} The incidence is now estimated at about one tenth of what it was, notwithstanding the enormously greater use of caustics which are sold in all grocery shops. Commercial lye preparations are about 95 per cent sodium hydroxide. Some of the drainpipe cleaners contain about the same percentage, along with gasifying chemicals (sulfuric acid in some preparations). The cleansing and washing powders contain from 8 to 50 per cent of caustic alkali, usually soda ash. Utensils used to measure or dissolve the powders are afterward used for drinking, without rinsing, and thus the residue of the powder remaining is swallowed in strong solution. At other times solutions of lye are drunk in mistake for water, coffee, or wine. Lye in skillets has been mistaken for gravy. "Household ammonia," "salts of tartar" (potassium carbonate), "washing soda" (sodium carbonate), mercuric chloride, and strong acids (most frequently sulfuric battery acid) are also the cause of chemical burns of the esophagus.

Pathology.—The effect of a swallowed caustic is usually exerted on the lips, tongue, and pharynx as well as on the esophagus. The levels most often and most severely affected are, in order of frequency (1) at the crossing of the left bronchus, (2) in the region of the cricopharyngeus, (3) at the hiatal level.^{1 4} Sodium hydroxide has great affinity for water and destroys moist tissue rapidly, but not instantly. If it remains in the esophagus, burns will be inflicted entirely around the periphery and will extend through the wall into the periesophageal tissues. Such burns are rapidly fatal. If the esophagus empties itself quickly burns are not so deep, and may not extend all around the periphery. This is important clinically. Healing of the ulcers that follow sloughing is slow, sometimes requiring months. Esophagoscopy examination at this time will show the thickened edge of the ulcer. The bed of the ulcer may be covered with grayish exudate or flabby granulations. At a later stage one edge may show beginning cicatrization. The sluggish healing results in building up a mass of cicatricial tissue usually occupying a segment of the periphery. A concentric stricture attached to the entire periphery of the esophageal wall occurs only in deep circumferential burns. Chronic esophag-

itis from stagnation and fermentation of foods may cause edema of the adjacent, noncicatricial area and may occlude the lumen of the stricture by edema, if long continued it may add to the building up of scar tissue. As with all other tubular channels cicatricial contraction results in stricture, and strictures are prone to become occluded.

Diagnosis—The diagnosis of chemical burns at the time of the accident is usually made on the statement of the patient or the parents as to the swallowing incident, and on the evidence of burns about the lips and mouth. The mouth is usually filled and overflowing with glairy saliva allowed to accumulate because of the dread of the pain of swallowing.¹⁻⁴ Sometimes, however, chemical burns of the esophagus occur without visible burns of the lips, tongue, mouth, or pharynx. This clinical fact we have often observed. It may be due to the undoubtedly greater delicacy of the esophageal mucosa or to there happening to be a heavy protective coating of saliva in the mouth at the moment of the accident. In all cases the patient who has swallowed a caustic should have a diagnostic esophagoscopy, preferably about two or three weeks after the accident.

Prophylaxis—The prophylaxis is to be considered from two viewpoints—the prevention of the accident and the prevention of stenosis of the esophagus. An adequate 'Poison' label, as mentioned in connection with incidence, probably has accomplished 90 per cent of the prophylactic work. It remains to educate everyone to keep all lye and cleansing preparations out of reach of children. To prevent stenosis of the esophagus the patient must not be allowed to neglect treatment during the 'symptomless interval.' At worst, a swallowed string, if not a duodenal tube, must be worn to prevent the disaster of total atresia of the esophagus.

Treatment—The treatment of chemical burns of the esophagus at the time of the intake of the caustic should consist in neutralization—vinegar to neutralize caustic alkalis, alkalies, such as sodium bicarbonate, to neutralize caustic acids. Mercuric bichloride requires raw eggs or flour mixed with water or milk. Now that the labeling of containers of caustics with simple antidotes has resulted from enforcement of the Federal Caustic Act, first aid treatment is sometimes applied in time.³ After the emergency in a child who has swallowed lye the next problem in most cases is that of avoiding de-

hydration. The copious flow of glairy saliva is retained and fills the mouth because of the dread of the pain on swallowing. In children this saliva also obstructs deglutition. It must be aspirated. As soon as the saliva has been aspirated a pinch of bismuth subcarbonate, say about 15 grains (1 gm.), is sprinkled on the tongue and washed down with water instilled into the mouth with a dropper, the child being dorsally recumbent. The local application of bismuth is also the best possible treatment for the burns about the mouth. The bismuth and water treatment should be repeated every half hour for three or four times, then about every three or four hours for a day or two, less often after that. After the first few days a good way to give the bismuth is in the form of a bismuth marshmallow (qv). Water must be regularly given in specified quantity according to the age of the child. Within two or three weeks diagnostic esophagoscopy should be done and thereafter a small esophagoscope should be passed about once every week or ten days to prevent formation of a stricture. Local applications to the corrosive esophagitis, whether ulcerated or not, is not necessary, the bismuth marshmallow is enough, but the esophagoscopies are necessary for maintaining a lumen and for guarding against stagnation of food. If this treatment is not carried out, usually the subsequent course in these cases, after the acute stage is over, consists in no trouble in swallowing for a few weeks (this is the delusive "symptomless interval"), then a slowly developing difficulty in getting solids down. Before long a hastily swallowed ill masticated bolus lodges and suddenly occludes the small remaining lumen. The child may be brought to the hospital for the removal of the bolus, this is readily done with forceps used through the esophagoscope. In many cases, however, the bolus passes on, and swallowing, favored by more careful mastication, is not troublesome for a time. Alternating occlusion and relief occur at shortening intervals. There comes a time when there is too long a wait for the bolus to pass on and the child is brought in dehydrated and acidotic. If old enough the child will be begging for water, if younger the baby will be moaning and the dehydration will be evident from the dry parched lips and tongue, the hollow eyes, and the dry skin. The statement of the relatives as to fluid intake is unreliable, the child may have swallowed large quantities of water and re-

gurgitated it all. No time is to be lost. Fluid must be introduced intraperitoneally or intravenously, or by hypodermoclysis. If the urgency is not extreme the colonic drip method is the best. The sucking of ice may be comforting. As soon as the child is out of danger from dehydration, esophagoscopy should be done. If a foreign body is corking a stricture it can be readily removed with forward-grasping esophagoscopic forceps. The child will then be able to swallow liquids, and a carefully-strained perfectly-balanced liquid diet with excess of vitamins should be given. As soon as the child's condition is improved *treatment of the cicatricial stricture* may be undertaken. This usually requires *esophagoscopic bougienage*. Bougienage is never done blindly. The lumen through the stricture is found with filiform silk-woven esophagoscopic bougies (Fig. 522) and dilated by slow progression of increasing sizes of these bougies. This requires utmost care and gentleness. It must always be borne in mind that the periphery of the stricture is not usually cicatricial. The part that is normal will split if over-stretched suddenly, whereas the scar is tough. Yet it is this normal segment of the periphery that will yield best to dilatation and the cure will be permanent, but it must be stretched gradually through a period of months to avoid fatal rupture. When sufficient lumen is obtained for its passage, the smallest size of duodenal feeding tube may be inserted, if swallowing is intermittent and unsatisfactory. The feeding tube is introduced through the esophagoscope, under guidance of the eye, a plain steel stilet without handle being used to give control of the limp tube. After it is introduced the tube is held in place by the stilet while the esophagoscope is withdrawn. The proximal end of the feeding tube is brought out through the nose and fastened to the cheek with an adhesive strip. Specific instructions as to quantity, formula, and timing of tubal feedings must be given to prevent avitaminosis, malnutrition, imbalance, and acidosis. Milk and eggs are not enough, fruit juices, sprouted vegetables, and excess of vitamins must be added. Water is forced through the tube after each feeding to prevent clogging of the tube. The total quantity of water must be specified and more should be given if the child is thirsty. Further treatment of the stricture is undertaken as soon as the child has recovered from the effects of dehydration and starvation. In most cases, how-

ever, this temporary wearing of the small-size duodenal feeding tube is unnecessary. Sufficient lumen is obtained at the first esophagoscopy, by removal of curds and food particles, to permit the swallowing of water and the just-mentioned strained foods and fruit juices. The esophagoscopic bougienage is continued at biweekly sittings, gradually stepping up the sizes of the bougies one at a time until the stricture has a lumen of about the normal diameter of the esophagus. This would be about size 24 French for a five-year old child. A patient of this age can then be taught to swallow a stomach tube of about this size. He is cautioned against pushing on the tube. He must be led to be proud of this "sword swallowing act," which inspires awe in other children witnessing it. Doing this once a week will keep the lumen open, so that anything can be swallowed, and the lumen of the stricture gets larger and larger as the child grows. The normal segment yields and enlarges until the cicatricial segment becomes a scar on the sidewall. Although a five-year old patient has been taken for illustration, children as young as two years can be taught the swallowing act. Younger patients with stricture are rare. If one is encountered the dilated lumen can be maintained by occasional passage of the esophagoscope until the child is old enough to be taught the swallowing act. It is safe to tell dependable parents that the swallowing act should be kept up for a year or two at weekly intervals, and that, if at any time the patient cannot swallow the stomach tube to the mark, the patient must be brought into the hospital at once for esophagoscopic examination and treatment. After a good lumen with scar on the side wall is obtained the patient may be discharged as cured. A print of a radiopaque roentgenogram should be filed for comparison in later years.

For clearness, the treatment of a child has been described in the foregoing paragraphs. Adults also get strictures from swallowing lye accidentally, during intoxication or with suicidal intent. They also are afflicted with cicatricial strictures due to trauma, endo esophageal or penetrating from the outside, also from various ulcerative diseases previously described. The methods of treatment are the same, but much easier, especially because larger esophagoscopes can be used. Even in the adult, however, the esophagus is intolerant, surgically,¹ the walls are thin and the mortality rate of perforation

is extremely high. With proper care and patience perforation is exceedingly rare.

Prognosis.—Any patient having any kind of lumen, however small, through a cicatricial stenosis of the esophagus can be cured by dilatation, with perfect restoration of function and with little danger to life. If the stricture is single and the entire circumference is not cicatricial the time required is only a few months. If the strictures are multiple and eccentric to each other, or if the entire periphery of the lumen is cicatricial, many months of patient careful treatment will be needed. Many patients have been thus cured. We have an interesting series of radiopaque roentgenograms taken in adult life, showing no defect in the esophageal shadow, to compare with childhood roentgenograms of the same patient made as many as twenty-four years previously, showing tight cicatricial strictures. In some cases the childhood condition was one of total atresia. A number of these patients are serving overseas in World War II.

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GASTROSTOMY

The methods of treating cicatricial stenosis of the esophagus described in foregoing pages require the skill of an experienced esophagoscopist. When a starving, dehydrated, acidotic child, unable to swallow water, must be treated

where no esophagoscopist is available a gastrostomy is required. Before the operation, water should be gotten into the circulation by the best available methods, in order to lessen the risk of operation. If there is time, colonic drip is best, but hypodermoclysis by intraperitoneal or intravenous methods may be used. After gastrostomy the same schedule of tubal feeding described under "Treatment" in "Burns of the Esophagus" must be enforced. Constant vigilance is required to prevent dehydration, avitaminosis, and malnutrition from laxity in feeding. The next great danger after recovery from gastrostomy is *total atresia of the esophagus*. This must be prevented by having the patient wear an endless string to maintain at least a fistulous passage. Any lumen, no matter how small, tortuous, or fistulous, can be dilated safely, but in total atresia a perforation must be made to connect the upper and



Fig. 530.—Retrograde bougie (Tucker) for the treatment of cicatricial esophageal stenosis in patients who have a gastric fistula.

lower esophageal lumina. We have done this many times successfully but the procedure is not free from risk of false passage and fatal mediastinitis. Two esophagoscopists cooperate, one working through the mouth, the other using a small esophagoscope through the gastrostomic fistula. Biplane fluoroscopic guidance is essential. The gap remaining, when the two esophagoscopes are insinuated as far as they will go, is perforated with the smallest opaque filiform (Fig. 522), either from above or below. The filiform is drawn out, grasped with forceps, and introduced from the opposite direction. One end of a 2-yard length of no. 14 braided silk is made fast to it, around the steel stem, above the filiform, by a clove hitch. Then the opaque filiform is withdrawn all the way out, the string is detached from it and tied to the other end of the braided silk to leave in an endless silk string. The dilatation of the stenosis is begun by retrograde methods.

Retrograde Dilatation of Cicatricial Stenosis.—In patients with cicatricial stenosis in whom gastrostomy has been done, retrograde bougienage is the best treatment. The retrograde

bougies (Fig 530) are pulled up from below by means of a swallowed string. About three gradually increasing sizes are used at each treatment. The treatments are repeated at intervals of a few days usually about twice a week. Between seances the patient wears an endless string through the nose, esophagus, stomach and fistula, a fresh string being dragged in for renewal at each seance. Even babies can be induced to swallow a string by a skillful tactful nurse who is patient and fond of children. Our nurses have rarely failed. Smearing the string with candy helps. A 2 yard length of no. 14 braided silk is wound on a folded paper

ach and gastrostomy fistula. The patient is now ready for retrograde bougienage.

For retrograde treatment of cicatricial stricture the treatment table is set up as shown in Figure 531.

The procedure is as follows:

- 1 The endless string is cut near the fistula.
- 2 The fistular end is attached to the long loop of braided silk to which is attached the train of three bougies.
- 3 The train of bougies is drawn into the fistula and through the fatal esophagus upward into the esophageal stricture. In drawing the bougies up the tract on is made as al by putting the index finger back into the pharynx doing the traction on the bight of the string.

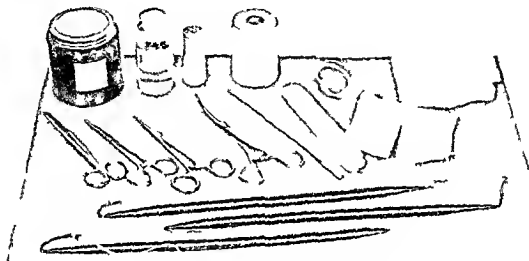


Fig 531—Layout for retrograde treatment of cicatricial stenosis of the esophagus in patients who have a gastrostomic fistula. Left to right back row: petrolatum, benzene for removing adhesive, spool of braided silk, roll of gauze. Middle row: scissors, 3 hemostats, pillar retractor, wooden tongue-depressor, gauze. Front row: train of three progressive sizes of retrograde bougies with long (60 cm) leader of doubled braided silk.

bobbin pinned to the child's waist to be unwound from time to time as the swallowed end passes downward. In the course of a few days the swallowed end will have passed through the stomach into the duodenum. The bight of the string stretched across the stomach from the hiatus to the cardia is readily caught and fished out with the pillar retractor. The lower end is withdrawn from the duodenum and is tied to the end unwound from the bobbin. (If the string has passed too far into the intestine to be withdrawn it may be cut off and the intestinal part allowed to pass on.) This makes a continuous string through mouth, esophagus, stom-

ach and gastrostomy fistula. The patient is now ready for retrograde bougienage.

For retrograde treatment of cicatricial stricture the treatment table is set up as shown in Figure 531.

The procedure is as follows:

- 4 The first two sizes are each allowed to rest a few moments in the grasp of the stricture. The third and largest bougie is allowed to remain for a few minutes or longer according to the tolerance of the patient.
- 5 The entire train of bougies is withdrawn from the mouth.
- 6 The last bougie is detached from the upper end of the new string, which is then brought through the nose and tied to the fistular end, making the new string endless and fit to be worn until the next seance.
- 7 The method of bringing the upper end of the new string from the mouth through the nasopharynx and

nose is this: One end of a small size (no. 12) retrograde bougie is passed back through the nose far enough to be seen and grasped in the pharynx. It is drawn out the mouth far enough to have the end of the esophageal string attached to it. Withdrawing the bougie from the nose brings with it the end of the new string.

At the first few seances great care is taken to avoid alarming the child. The small-size bougie is used so it will come up through easily. In some cases the child is taught to help pull it up himself, the operator's index finger being used in the fauces to maintain axial traction and prevent the string from cutting into the tongue when the child pulls on the thread. He must be taught not to jerk, or pull hard or fast. Most children become proud of the feat. Before the bougies are passed they are lubricated with a fold of gauze dipped in petrolatum. After the seance this lubricant is removed from the bougies by thorough washing with soap and warm water. The treatments are best given by nurses. Their gentle handling and persuasive way with children soon convince the child that the treatments are not operations. Like the esophagoscopy bougienage the procedure is totally free from mortality, provided the same precautions are taken as to gentleness and avoidance of overstretching the normal segment of the stricture. A number of months are required before a stricture of small lumen can be enlarged to the stage at which a child can be taught to swallow a stomach tube as described in connection with esophagoscopy bougienage.

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ULCER OF THE ESOPHAGUS

An ulcer is a superficial area of molecular death. The area is limited. General surgical classifications are numerous and confusing. As encountered in the esophagus there are two main classes—the malignant and the infective. There are two classes of infections—the specific and the nonspecific in origin. All ulcers in this region become secondarily invaded by the common pyogenic invaders, but the primary character remains throughout the course of the disease the most important factor from clinical viewpoint.

A *nonspecific ulcer* is a patch from which the epithelium is lost. It may start as a gradual disintegration of the epithelial cells that has been loosely called an "erosion." The epithelial cells may be disarranged sufficiently to permit penetration of infection into the subepithelial tissues, or the epithelial layer may become suddenly necrotic over a limited area. In either case, once infection gets beneath the epithelial barrier infective inflammation produces the characteristic features of an ulcer—a bed of granulation tissue surrounded by a border of inflammatory tissue that retains its epithelial covering. The area and the depth may be increased by extension of molecular death. If the ulcer remains superficial it will leave no scar on healing. When the deeper layers of the mucosa and the submucosa are penetrated scarring is inevitable. Embryonic tissue becomes vascularized into granulation tissue, which in healing becomes fibrous tissue, and this scar tissue contracts. Contraction in the walls of a tubal organ like the esophagus always tends toward stricture of the lumen. The causes of nonspecific ulcers are pyogenic organisms and a localized vulnerable area. This whole subject has been studied by Harris P. Mosher. (See under "Fibrosis of the Esophagus.")

Specific ulcers of the esophagus are due to specific organisms such as those of Vincent's infection, *Odium albicans*, *Vibrio tonsillaris*, *Blastomyces dermatitidis*, *Mycobacterium tuberculosis*, *Spirochaeta pallida*. These infections may penetrate the epithelium or invade by way of the blood vessels or lymph channels, the involved area breaking down and forming a specific ulcerogranuloma. Suppurative tuberculous glands break through in this way. Any abscess opening into the esophagus becomes

an ulcer, practically, the bed of an ulcer is the wall of an abscess on a free surface. The diagnosis can be made by smears and cultures from esophagoscopically obtained specimens. This alone should be depended upon in the shallow clean-cut ulcers such as that of muguet, which is usually due to *Oidium albicans*, and similar lesions due to monilia, saccharomycetes, or other fungi. In all the others there is usually a thickened granulomatous border from which a specimen of tissue for biopsy may be taken without danger of perforation. In cases of ulcer following the swallowing of caustics, a smear is sufficient. The diagnosis of *malignant ulcer* is by biopsy, which is always conclusive if the specimen is properly taken (see under "Malignant Tumors of the Esophagus").

The treatment of the different varieties of ulceration is given under the respective headings. The best local treatment for any ulcer of the esophagus and its accompanying *acute* or *chronic esophagitis* is the bismuth marshmallow. The ordinary confectioner's marshmallow is cut in four pieces, once across each way, the cut sides are pressed into powdered bismuth subnitrate, and they are then dispensed in a box containing powdered bismuth subnitrate, some of which adheres to each piece. If the marshmallows are fresh and the freshly cut sides are well pressed into the powder each one will carry about 5 grains (0.3 gm.) or less, and can be taken every third hour or oftener by a child. As the action is local and qualitative, not quantitative, the dosage is unimportant. The bismuth adheres to the ulcer and forms a nontoxic, local, antiseptic dressing. It adheres also to inflammatory mucosa. To avoid alarm, parents should be informed of the blackish discoloration of the tongue by the sulfiding of the adherent bismuth. The bismuth marshmallows are for children. Adults usually dump the contents of a powder paper, ordinarily 15 grains (1 gm.), dry, on the tongue and swallow it with saliva. A capsule may be used for dispensing if the patient can be trusted to let it dissolve in the mouth before swallowing it. For *peptic ulcer* at the lower end of the esophagus, posture is helpful. Sleeping with head and thorax high minimizes hiatal return flow of acid gastric juice.

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TUBERCULOSIS OF THE ESOPHAGUS

Tuberculosis, like some other diseases, is so peculiarly rare as a primary lesion in the esophagus, in comparison to the larynx, for example, as to raise an interesting question of the reason for the relative immunity. Tuberculosis of the larynx (*qv*), extending down into the hypopharynx, almost always stops short of the cervical esophagus. That the esophageal wall is not invulnerable is shown by the many cases of extension by continuity from tuberculous nodes near the root of the left lung.

The symptoms are dysphagia and odynophagia, neither is often severe, usually the patient has a dull feeling of the bolus of solid food passing a tender spot. Cough is usually present, it may be from the passing of food or liquids through an esophagobronchial fistula, from obstructive overflow, or from the accompanying tuberculous lesion in the tracheobronchial tree.¹ In most cases the esophageal symptoms are entirely obscured by symptoms from the tracheobronchial or laryngeal lesions. In many instances the diameter of the esophagus is found at autopsy retracted by the cicatricial contraction of a healed tuberculous lymph node. Such cases are observed esophagoscopically during life.¹

The diagnosis of an esophageal tuberculous granuloma, the usual lesion, can be made only by biopsy, the specimen being taken with the ball forceps used through the esophagoscope. The specimen requires examination by smears, the microscopic examination of tissue for histologic evidence of tuberculous processes, and

also examination of specially-stained specimens to show the *Mycobacterium tuberculosis*

Local treatment is not indicated when the esophageal lesion accompanies bronchopulmonary tuberculosis, as it usually does, unless the esophageal lesion is so obstructive as to interfere with that essential element of treatment of all tuberculous disease, nutrition. In such a case dilatational esophagoscopic bougienage is indicated, whether the esophageal lesion is active or cicatricial, or both. Whether stenotic or not, the treatment indicated for esophageal tuberculosis is the general antituberculous regimen of the modern system of treatment of bronchopulmonary tuberculosis.

The prognosis of the tuberculous esophageal lesion is not bad in itself, but usually it is that of the associated lesions. In one of our cases there was a good recovery, the patient was known to be still in good health four years later.

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tients to have a serologic test, but in case of a patient with any abnormality in swallowing a positive reaction is never taken as a conclusive diagnosis of syphilis of the esophagus. Esophagoscopically removed specimens of tissue examined histologically show the significant perivascular and other changes of syphilis, and, most important, they can determine the presence or absence of histologic evidence of cancer. As in late lesions elsewhere, spirochetes are difficult to demonstrate in tissue. The esophagoscopically observed behavior of an esophageal syphilitic lesion under antisyphilitic treatment is good supportive evidence, but the administration of such treatment as a test is inadvisable until at least a tentative diagnosis of syphilis is made.

The treatment of esophageal syphilis is systemic, the details are given in connection with the subject of "Syphilis" discussed under "Dermatoses of the External Nose." Locally it is advisable to cover the ulcer of breaking down gumma with a coating of bismuth subnitrate, which can easily be done by giving the powder dry on the tongue in doses of 15 grains (1 gm) after every meal and again at bedtime (see under "Ulcer of the Esophagus.") The bismuth in this form is not absorbed and has no spirocheticidal effect. It adheres to the ulcerated surface and has an antiseptic effect on mixed pyogenic infections. Treatment of syphilitic cicatricial stenosis is by the silk-woven esophagoscopic bougie (Fig 522), under guidance of the eye. The prognosis of esophageal syphilis is good if the diagnosis is made early and if the patient cooperates faithfully in the treatment. Strictures can be cured by patient, careful esophagoscopic bougienage.

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SYPHILIS OF THE ESOPHAGUS

Syphilis of the esophagus is a rather rare form of syphilis but we have seen a number of cases. It may occur as an early mucous plaque, a late chronic leukoplakia, a gumma, an ulcer, a granuloma, a cicatrix, an aneurysmal compressive stenosis, or as a neurosyphilitic manifestation. The esophagoscopic appearances have been illustrated in color 1 2 3. As in any other lesion in the esophagus the secondary pyogenic infections alter the appearances of ulcerations. It is the invariable rule for all pa-

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MYCOSES OF THE ESOPHAGUS

Actinomyces of the Esophagus—This disease is uncommon, it is certainly less frequent than mycotic infections of the tracheobronchial tree, though, in the esophagus, mycotic infections are overlooked in an even larger percentage of the cases. Farmers and cattlemen are most frequently exposed. The infective agent is the ray fungus, *Streptothrix actinomyces*, characterized by club shaped bodies in pinhead-sized nodules with abundant shedding of spores. In our cases the disease of the esophagus was secondary, other lesions were in the neck, the lower alveolar arch, in the lungs, and in the mediastinum. In one case the esophageal lesion was integral with the lesion in the left bronchus. The esophagoscopy appearances were those of small ulcerogranulomatous elevations with a tendency to ridge like formation. Small granules containing characteristic ray fungi were removed, along with granulomatous fragments, by means of the esophagoscopy ball forceps. Microscopic examination of such specimens is the only means of diagnosis of esophageal actinomyces, but it is always conclusive. Dysphagia is present in obstructive lesions, and there may be regurgitation. Earlier lesions produce only a slight sensation of a swallowed bolus passing a "sore place." Local treatment is not indicated unless the stenosis is sufficient to interfere with nutrition. In such a case dilatation is indicated. A nasal feeding tube may be necessary. General surgery is sometimes advisable for associated actinomycotic lesions. Potassium iodide is unquestionably beneficial, sometimes curative early in the disease. It should be commenced in small dosage, about 5 drops of a saturated solution, given with a tumblerful of water three times daily. Increase should not be more than a drop each day, and 30 drops to the dose would be the limit. The treatment can not be stopped because of iodism, in fact iodism must be maintained for efficient concentration. The prognosis is unfavorable, but not hopeless.

Blastomycosis of the Esophagus—Blastomycotic lesions are rare in the esophagus, but we have seen four cases. In all there was difficulty in swallowing due to involvement of the esophageal wall. The diagnosis was made by the microscopic examination of specimens esophagoscopically removed with the ball forceps. The specimens were examined unstained

because the blastomycetes are thus more readily seen. The esophagoscopy appearances were those of a thickening and rigidity of the esophageal wall, with granulomas on ulcerated beds in the stenotic lumen. It was from this ulcerogranulomatous lesion in each case that the specimens were esophagoscopically taken. Dilatation was required to maintain sufficient lumen for swallowing. In one patient, an ulcerogranuloma was found in the left main and both right and left upper lobe bronchi. This patient died about a year later, apparently from the associated tuberculosis and mycosis. Permission for autopsy was unobtainable. The other three patients recovered fair health, with some narrowing of the healed esophagus. The treatment given was potassium iodide as mentioned in the previous paragraph. The prognosis of blastomycosis of the esophagus is probably better than in other mycoses in this region.

Sporotrichosis of the Esophagus—This is due to infection with *Sporotrichum schenki*. It occurs as a small abscess, or a few small abscesses, and is associated with multiple abscess elsewhere in the body, especially in the subcutaneous tissues, where it is known as "cutaneous sporotrichosis." The diagnosis is made by microscopic examination of fresh smears and the histologic examination of the chronic granulomatous walls of one of the abscesses. Syphilis and tuberculosis are always to be considered in differential diagnosis and as requiring treatment if associated. The treatment of sporotrichosis is by potassium iodide as advised for blastomycosis. It is a specific. Abscesses in the esophagus may leave a cicatricial stenosis requiring peroral esophagoscopy bougienage.

Muguet.—There are two clinical types of this disease, both apparently due to *Oidium albicans*, though other fungi are found. One type is the acute self limited aphthous stomatitis of young children, it is often called "thrush." The other type is the constantly recurring, slowly healing aphthous ulcer of older children and young adults. Both types occasionally occur, though rarely, in the larynx, hypopharynx, and esophagus. We have had occasion only once to do an esophagoscopy on a baby with thrush. He had a coin lodged in the esophagus. There were no lesions of thrush in the hypopharynx or esophagus. In the adult type the ulcerative lesions are seen as one or more isolated superficial punched out ulcers rarely over 3 mm in diameter. Initially the bed of the ulcer is white,

like the ulcer of thrush in babies, but in the course of a week or less, as the result of mixed secondary infections, the color of the bed is greenish, then it becomes yellowish in color, the surrounding mucosa at this later stage is red and swollen. Usually healing takes place in less than two weeks. The ulcers are tender to the touch of food, especially acid fruits and also vegetables, such as tomatoes. Though primarily and secondarily due to infection, there is a predisposing factor of avitaminosis and intestinal stasis. These form the basic indications for prophylaxis and treatment. The occurrence of successive crops can be stopped by the daily administration (preferably before the evening meal) of one quarter of the formula of cascara-compound pills. This dosage must be revised according to effect. Intermittent catharsis is a mistake. A quantity of green, leafy vegetables must be eaten every day, and an excess of mixed vitamins must be given daily. The chief symptom, pain on contact of food in swallowing, can be ameliorated by the administration of bismuth subnitrate, 15 grains (1 gm) dry on the tongue, immediately after each meal, and again three hours later. It will cap the ulcer with a protective antiseptic coating. In children the bismuth marshmallow is the best form of administration, and it is curative. The prognosis is good.

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COMPRESSION STENOSIS OF THE ESOPHAGUS

The esophagus may be narrowed by the pressure of any periesophageal disease or anomaly. The lesions most frequently found are (1) goiter, cervical or thoracic, (2) intra thoracic tumor, (3) aneurysm, (4) cardiac or aortic enlargement, (5) lymphadenopathies—(a) Hodgkin's disease, (b) leukemia, (c) syphilis, (d) tuberculosis, (e) infective adenitis, (f) non specific infective adenitis, (6) lordosis, (7) enlargement of the left hepatic lobe. Deviation or displacement may accompany the compression. Esophagoscopically, compression stenosis of the esophagus is manifested by a slitlike crevice which occupies the place of the lumen and does not open up readily before the advancing tube. The long axis of the slit is almost always at right angles to the compressive mass, if the

esophageal wall is uninvolved, as is usually the case in purely compressive conditions. The covering mucosa may be normal or it may show signs of chronic inflammation or erosion from stasis. Malignant compressions are characterized by their hardness when palpated with the tube. Associated pressure on the recurrent laryngeal nerve often makes laryngeal paralysis co-existent. The nature of the compressive mass outside the esophagus will require for its determination the aid of physical signs and roentgen ray studies as well as esophagoscopy examination. Compression by the enlarged left auricle has been observed at our clinic a number of times, and auricular enlargement does not contraindicate esophagoscopy. The presence of aneurysm is a distinct contraindication to esophagoscopy for diagnosis except in case of suspected foreign body.

Treatment of compression stenosis of the esophagus depends upon the nature of the compressive lesion, and is usually systemic except when the compression is sufficient to interfere with nutrition. This degree of stenosis is usually relievable by careful esophagoscopy dilatation and is free from danger except in case of aneurysm. In case of foreign body or food lodged in the lumen of an aneurysmal compression the intruder should be removed esophagoscopically without hesitation. We have had a number of such cases, and no complication arose in any of them. Intubation risks erosion. Gastrostomy is rarely required.

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BENIGN TUMORS OF THE ESOPHAGUS

A tumor that does not infiltrate neighboring tissues and is not likely to recur is customarily called "benign." As applied to the esophagus, "innocent" is not a proper synonym because benign tumors in this location have caused

death by asphyxia, respiratory paralysis, and hemorrhage. Statistics show a rare incidence of benign growths of the esophagus. Our clinical records show a number of cases in the course of many years, but as compared to benign growths in the larynx and pharynx they are rare. The cause of benign growths anywhere in the body is controversial, probably some of them are embryonal in origin, others are in part inflammatory. At our clinic we have observed cases of papilloma, adenoma, angioma, myoma, fibroma, lipoma, myxoma, edematous polyp, and cystic growths. Good results followed esophagoscopy removal. In six cases removal was necessary to relieve stenosis. This was accomplished with tissue forceps, used through the esophagoscope, and the growths did not recur.¹ Ellen J. Patterson reports a very interesting case of papilloma.² In one of our cases prolonged stasis from multiple obstructive papillomas resulted in cicatricial stricture requiring two years' esophagoscopy dilatation for cure.

If the tumor is single and small there may be no symptoms. Large tumors or large masses of multiple papillomas may cause dysphagia and regurgitation. A long pedicle may permit regurgitation and swallowing of the tumor.³ Bronchial symptoms may occur from overflow.⁴ Cough is frequent and may be (1) from overflow, (2) from pressure, (3) from the flopping of a pedunculated growth into the larynx, (4) from irritation of the laryngeal excitation center. Slowly growing tumors allow time for the normal portion of the esophageal wall gradually to dilate to compensate for encroachment of the tumor on the lumen. Esophagoscopy is the only means of diagnosis. The color of benign growths is pinkish or whitish except in case of angiomas and varices, these may be pink but more often are bluish or purplish. The tumors are usually pedunculated in form, but even if not the experienced eye will quickly differentiate a tumor from a fold. Biopsy is advisable and is free from danger if the precautions mentioned below are taken.

The best form of treatment is esophagoscopy removal. This is readily accomplished with the ball forceps (Fig. 522), if the growths are small and project into the esophageal lumen. The determination of the advisability of the removal of very large growths requires careful consideration of the particular case. The extreme thinness of the esophageal walls must be always in the mind of the esophagoscopist if he

would avoid disaster. Except in cases of a varicosity there is no danger in removing tumors, but a small nip taken out of the esophageal wall might lead to fatal complications. Strong traction on a fibrous pedicle would risk tearing the wall. Usually the radical removal of the base of a benign growth is contraindicated. Intrusion of an aneurysm must be excluded beforehand. Esophageal bleeding can be arrested by the administration of bismuth subnitrate (1 gm.) dry on the tongue every fifteen minutes. The bismuth marshmallow is best for children. The dissolving of ice in the mouth is also an excellent remedy. Banti's disease and varices are always to be thought of. We have seen a number of these cases at our clinic. The local condition had been mistaken for angiodysplasia for the removal of which the patient was referred.

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MALIGNANT TUMORS OF THE ESOPHAGUS

A malignant tumor of the esophagus may be defined as a new growth in the esophagus that infiltrates neighboring tissues, has a tendency to formation of metastases and to recurrence after removal. Cancer, though originally applied only to carcinoma, has come into general use as a synonym for any malignant growth.

As among diseases in general, a malignant tumor of the esophagus is uncommon, but in a busy broncho-esophagologic clinic it is a distressingly common malady. Though it is always overlooked early, its terminal symp-

toms are so characteristic that mortality statistics are fair criteria of incidence. In the year 1938 there were recorded in the United States 149,214 deaths from cancer, of which 2498 were recorded as of the esophagus. During that year there were 1,381,391 deaths from all causes.

Etiology.—Age is the only definitely proven causative factor. In men it usually develops during the fourth and fifth decades of life. In women cancer often occurs at an earlier age than in men, and tends to run a more protracted course, preceded in some cases by years of precancerous dysphagia. It is almost unknown in persons under twenty years of age.

Pathology.—Squamous-cell epithelioma is the most frequent type of neoplasm. In the lower third of the esophagus, adenocarcinoma is often found associated with a like lesion in the stomach. Sarcoma of the esophagus is relatively rare, but we have seen a number of cases.^{1 2} In one case a leiomyoma developed malignant activity with extensive metastases, myosarcomatous in type, and it proved fatal.³ The middle third of the esophagus is most frequently involved, the lower third, near the hiatal constriction, comes next in frequency. Cancer of the lower third of the esophagus preponderates in men, while cancer of the upper orifice is, curiously, more prevalent in women, in the proportion of 6 to 1. The lesion is usually single, but multiple lesions resulting from implantation metastases, have been observed.¹ Bronchoesophageal fistula from extension is not uncommon. The extension may be in either direction.

Symptoms.—Malignant disease of the esophagus is rarely seen early, because the vague indefinite sensations of the patient are either ignored by the practitioner or are brushed aside as neurotic or as a *globus hystericus*.^{1 4} The absence of pain and loss of weight is misleading. Dysphagia, the one common symptom of all esophageal disease, is often ignored by the patient until it becomes so marked as to prevent the taking of solid food, therefore, the onset may seem abrupt. Any well-masticated solid food can be swallowed through a lumen 5 mm in diameter. It cannot be too strongly emphasized that the dysphagia is intermittent in cancer of the esophagus. The inability to maintain the nutrition is evidenced by loss of weight and the rapid development of cachexia. When the stenosis becomes so severe that the fluid intake

is limited, rapid decline occurs from water starvation, and unbalanced diet. In rare cases a sloughing type of cancer may cause little dysphagia until late in the disease, but such patients always notice and complain of some abnormality in swallowing. Cancer is unsuspected until late in such cases. Pain is usually a late symptom of the disease. It may be of an aching character and referred to the vertebral region or to the neck, or it may only accompany the act of swallowing. Blood streaked, regurgitated material, and the presence of odor, are late manifestations of ulceration and secondary infection. In some cases, constant oozing of blood from the ulcerated area adds greatly to the cachexia. If the recurrent laryngeal nerves are involved, unilateral or bilateral paralysis of the larynx may complicate the symptoms by cough, dyspnea, and hoarseness. The same symptoms may be due to direct neoplastic extension, or to inspiration of food and secretion that overflows into the larynx because of extension. Hiccough may be troublesome when the lesion is at the lower end of the esophagus.

Diagnosis.—The foregoing mention of symptoms is for the purpose of giving the reader a general picture of the disease. For diagnosis there are only two diagnostic methods worthy of a moment's consideration, namely, the roentgen ray and esophagoscopy. By these two methods the diagnosis can be made with absolute certainty and at the very incipency of the disease. The present appalling mortality (nearly 100 per cent) is due to reliance on inferential means (the history of the case and blind bougienage) which always fail in revealing abnormality in the early stage of the disease.^{2 4 5} When all patients mentioning the slightest abnormal sensation in swallowing are endoscopically studied, precancerous lesions may be discovered and treated, and the limited cancer of the early stages may be afforded surgical treatment while there is still hope of complete removal. Moreover, it cannot be too strongly emphasized that early diagnosis is essential for prolongation of the patient's life by palliative measures. It is inhuman and unscientific to say that, because curative treatment is as yet ineffectual, there is no use making an early diagnosis.

The possibility of syphilitic or tuberculous ulceration of the esophagus is to be eliminated by suitable tests, supplemented by biopsy. Also the possibility of aneurysm of the aorta must

be excluded in all cases of dysphagia. The dilated aorta may be the sole cause of the condition, and the dilatation contraindicates esophagoscopy unless there are clear indications for doing it, such as the presence of a foreign body. Intermittent stenosis of the esophagus may or may not be malignant. Long duration of symptoms does not exclude cancer. In nearly all patients with cancer of the esophagus who have come to our clinic the condition has at some previous time been diagnosed as simply spasmodic, and many of these patients had been elsewhere treated for a year or more under this erroneous diagnosis. Esophagoscopy and removal of a specimen for biopsy render the diagnosis certain. This is done with the ball forceps (Fig. 522). It is very unwise to bite through normal mucosa for the purpose of taking a specimen from a periesophageal growth. Fungations and polypoid protuberances afford safe opportunities for the removal of specimens of tissue. In all our experience we have never seen complications of any kind follow the taking of a specimen of tissue for biopsy and in no instance was a histologic diagnosis of cancer incorrect. Statistics collected in 1934 showed that the same results are obtained by our pupils.⁶ Some of the most gratifying experiences in this work were the hundreds of instances in which we were able to bring back to a happy life a patient in the agonies of despair caused by an erroneous diagnosis of cancer made by inference. Conversely many other patients sent in with a diagnosis of "spasm" were found to have a hopelessly inoperable cancer. They were beyond efficient palliative treatment. A common error of inferential diagnosis is to exclude cancer because of long duration of symptoms. Such symptoms may be present for twenty years, due to other causes. Both classes of case illustrate the folly of attempting to make a diagnosis of esophageal disease without looking at the esophagus. Passing the bougie blindly is dangerous, inconclusive, and obsolete in esophageal diagnosis.

The esophagoscopic appearances of malignant disease of the esophagus,⁷ 5 varying with the stage and site of origin of the growth, we found to be as follows: (1) Submucosal infiltration covered by perfectly normal membrane, usually associated with more or less bulging of the esophageal wall, and often with hardness and infiltration. (2) Leukoplakia. (3) Ulcer projecting but little above the surface of the edges

(4) Rounded, nodular, grouped masses. They may be in mulberry-like form, dark or light red or pink in color. (5) Polypoid masses. (6) Cauliflower fungations. Blood may tint the image of any of the lesions enumerated except those of the first group (Fig. 346).

In considering the esophagoscopic appearances of cancer, it is necessary to remember that after ulceration has set in, the cancerous process usually has engrafted upon it, and upon its neighborhood, the results of inflammation due to the mixed infections. Cancer invading the wall from without may for a long time be covered with perfectly normal mucous membrane. The significant signs at this early stage are (1) absence of one or more of the normal radial creases between the folds, (2) asymmetry of the inspiratory enlargement of lumen, (3) sensation of hardness of the wall on palpation with the tube, (4) the involved wall will not readily be made to wrinkle when pushed upon with the tube mouth. In all the later forms of lesions the two characteristics are (a) the readiness with which oozing of blood occurs, and (b) the sense of rigidity, or fixation, of the involved areas as palpated with the esophagoscope, in contrast to the normally supple esophageal wall. Esophageal dilatation above a malignant lesion is rarely great, because the stenosis is seldom severely obstructive until late in the course of the disease. Sometimes cancer is secondary to a dilatation. Of most importance in endoscopic diagnosis is the education of the eye to the normal appearances and the variation within the normal. The pale mucosa of the anemic patient is very different from the red engorged mucosa of the plethoric patient. Transdiaphragmatic hernia of the stomach was the lesion in a number of patients coming to the clinic with a supposed cancer. Their needless mental distress was deplorable. The peptic ulcer so often present in transhiatal gastric hernia is flat and superficial, it heals and reappears in new locations, and scarring of the mucosa is often present.⁴ This latter disease is described in a separate section.

Treatment.—Early cancer of the first few centimeters of the cervical esophagus is reachable by lateral pharyngectomy (q.v.). The present nearly 100 per cent mortality in cancer of the thoracic esophagus will be lowered and a certain percentage of surgical cures will be obtained when patients with vague esophageal symptoms are given the benefit of early esoph-

agoscopic study ² The relief or circumvention of the dysphagia requires early measures to prevent food and water starvation. It is not generally realized that early diagnosis is just as essential for best results in palliative treatment as for attempt at cure. It is easier to retain weight than to regain lost weight. With early esophagoscopic diagnosis and a proper diet the patient can be maintained for a year or two in a fair state of general health and activity. In an unmanaged diet there always lurks the danger of acidosis, depression, melancholy, or loss of morale, conditions which result in irretrievable loss of vitality. Fatigue and worry should be avoided, plenty of sleep insisted upon. Elimination by bowels, skin, and kidneys should be gently but continuously increased, and, above all else, a careful supervision of the diet should be continuously maintained.

Unless guarded against, in nearly all patients, as swallowing becomes difficult two errors will occur: (1) improper or improperly prepared food will lodge, causing esophagitis and swelling that increase the obstruction, (2) the patient will take a little clear broth or coffee or tea, sometimes irregularly supplemented with raw eggs or milk. An abundance of vegetables and raw fruits is essential, of course, they must be sieved or strained so as not to lodge. The mesh of the sieve should be about 1 mm. and before using it the food should be passed through a "seprosil." Any food that can be pressed through a wire mesh of the size mentioned will go through a very small fistulous lumen or the smallest tube. We have found the appliance called a "seprosil" (manufactured by the American Utensils Company, LaSalle Street, Chicago) invaluable, in taking out the obstructive fibrous elements, in preparation of food for patients with obstructive esophageal lesions. It is invaluable also in preparing food for tubal feeding. Milk should not be given at or near the same time as curd producing foods, because curds will obstruct a small lumen. All foods should be thoroughly masticated and especially insalivated, even milk should be chewed *ie*, moved about in the mouth until mixed with saliva. The importance of saliva in nutritive processes cannot be overestimated, but it is generally overlooked ⁷

If the exactly calculated and measured quantity of the perfectly balanced diet, always containing an excess of mixed vitamins, can no longer be swallowed the cancerous lumen may

be dilated by gentle and careful *esophagoscopic bougienage* (q.v.) When this fails a feeding tube may be worn through the oesophagus indefinitely. If the patient prefers, a gastrostomy may be done. It should not be postponed until the patient becomes a poor risk. The poor statistic showing of gastrostomy is due to postponement and to imperfect dietary management afterward. Curative treatment has yielded relatively good results only in cases of early esophagoscopic diagnosis. The future outlook is better because of recognition of the necessity for prompt esophagoscopy in every case of the slightest abnormality in swallowing, and also because of technical advances in esophagectomy ⁸ Such a formidable procedure as transthoracic esophagectomy is only justified when biopsy has proved the cancer is present. There is no excuse for error in diagnosis, nor is there need for exploratory diagnosis, in any case in which the esophageal mucosa is involved.

There is still hope of the future discovery of a nonsurgical cure for cancer. Radium for esophageal cancer has so far been disappointing in our experience. If the dosage is sufficient to exert any effect, the patient grows rapidly worse from toxemia, his life is shortened and made more miserable. This is in marked contrast to the very satisfactory effects of radium obtained in other regions of the body. The same is true of roentgen ray therapy. The best palliative irradiation reported is by esophagoscopic implantation of radon seeds by the method of V. E. Negus.

Prognosis—The mortality of cancer of the thoracic esophagus is close to 100 per cent. With the overlooking of the disease and the usual deplorably late diagnosis the patient seldom lives more than a few months after the diagnosis is made. With an early esophagoscopic diagnosis and proper management of the feeding of a properly balanced diet the patient can have two years of rather comfortable and certainly useful existence. The prognosis following operative treatment in case of high cervical involvement is somewhat less unfavorable but the number of cases is as yet insufficient for statistical evaluation of results.

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the esophageal lumen is undiminished the patient cannot swallow Dehydration must be prevented and a perfectly-balanced and ample diet must be maintained This is accomplished by use of a small diameter feeding tube worn through the nose It is of utmost importance that the feedings be carefully formulated and strictly timed, as mentioned under "Paralysis in Relation to the Esophagus" The feedings should contain an excess of mixed vitamins

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DIPHTHERIA OF THE ESOPHAGUS

Infection of the esophageal mucosa with *Corynebacterium diphtheriae* is relatively rare We often thought it remarkable in the days when control by antitoxin was unknown that a virulent diphtheritic process extending downward would stop short, usually at the hypopharynx, but almost always at the cricopharyngeus The observations were made in the direct examination and aspiration of the larynx and trachea in diphtheritic children At the autopsies, much more frequent than now, the esophagus was found involved in only a little over 1 per cent of the cases The larynx and trachea were involved in 18 per cent of the cases in the same period In esophageal cases, vomiting of shreds of membrane occurred in some instances Older children complained of aching in the back and back of the sternum, also of nausea They always sank rapidly into profound toxic stupor and died within less than twelve hours Such cases occur today only when, for some reason, no antitoxin is given until too late Post-diphtheritic paralysis is still not uncommon It is toxic and not related to diphtheria of the esophagus The paralysis affects the constrictors of the pharynx The prognosis is good if there is a prompt realization that even though

PARALYSES IN RELATION TO THE ESOPHAGUS

The paralyses related to the esophagus are those affecting its primary function, *deglutition*, and its secondary function, *drainage* The latter serves to prevent overflow into the larynx and to carry down into the stomach for destruction the bacteria generated and collected in the mouth, nasal passages, nasopharynx, pharynx, larynx, and lungs Drainage is partly passive but is largely dependent on the active function of swallowing

The Physiology of Deglutition—The complex, coordinate, muscular sequence concerned in the act of deglutition may, for simplicity of fundamental presentation, be divided into the three stages concerned in swallowing solid food (1) passage of a bolus from the mouth to the pharynx, (2) passage of the bolus through the pharynx into the esophagus, (3) passage of the bolus through the esophagus to the stomach In the first stage the food on the upper surface of the tongue is roughly formed into a bolus which is raised to the roof of the mouth by the heaving up of the oral floor composed of the two mylohyoid muscles As the tip of the tongue strikes the roof of the mouth the tongue curls and heaves backward with the aid of the hyoglossus, styloglossus, and pharyngopalatinus muscles, pushing the bolus through the nar-

rowed isthmus of the fauces into the grasp of the pharyngeal constrictors. These powerful muscles compress the bolus and force it through the first closed gateway, where the cricopharyngeus pinches shut the mouth of the esophagus (Fig. 532). This gate coordinately opens a fraction of a second before the bolus arrives. The throwing of the bolus into the esophagus ends the voluntary part of deglutition. Thence to the stomach the bolus passes not by gravity but by peristalsis, which consists in the coordinated

passes through promptly. The afferent impulses concerned in the reflex arc of the peristaltic wave are discussed in a following paragraph entitled "Sensory Paralysis of the Esophagus." The foregoing description refers to swallowing a bolus of solid food which distends the esophagus. Liquids do not normally distend the esophagus and apparently are projected through to the hiatal gate, by the hulklike contraction of the powerful pharyngeal constrictors, in a fraction of a second, without any aid of peristalsis,

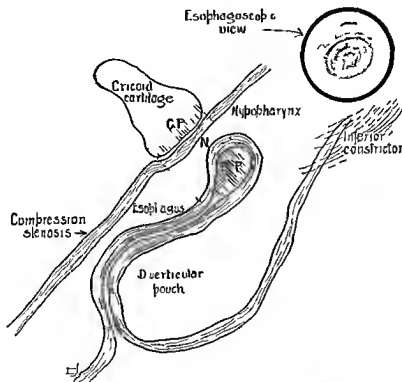


Fig. 532.—Schematic illustration of the function of deglutition, and, incidentally, the influence of this function on the formation of pharyngeal diverticulum. The cricopharyngeus muscle CP normally pulls the cricoid cartilage back against the spine maintaining by pinching a tonic closure that is constant, except for momentary opening by coordinate relaxation at the approach of a bolus of food propelled by the constrictors of the pharynx. On rare occasion abnormal failure to open puts undue pressure on the weak triangle in the support of the hypopharyngeal wall and a hernia known as a 'hypopharyngeal diverticulum' results. Later, compression stenosis of a large sac may help to increase the size of the sac.

muscular contraction behind the bolus to move it onward and a muscular relaxation ahead of the bolus. These movements progress in a wavelike manner under the control of the sympathetic autonomic system coordinated with the vagal antagonistic system. When the bolus reaches the second gateway, where the esophagus is pinched shut at the hiatus, the bolus has to wait until the gate opens. This waiting period varies within normal limits from a fraction of a second to say six or seven seconds. As soon as the gate opens the bolus

though sometimes the peristaltic wave coming slowly after will arrive in time for the opening of the gate, sometimes it is as much as six or seven seconds later.

The just given, simplified statement of deglutition concerns the movement of food from mouth to stomach. There is coordinated with this movement another mechanism which is concerned with shutting side gates to keep the food from going where it should not, namely, into the nasopharynx and into the larynx. This accessory system also must be considered in

connection with paralysis. The closure of the larynx has been described in connection with the deglutitory function of the larynx. The closure of the nasopharynx is accomplished by contraction of the levator palati, tensor palati, palatoglossus, and palatopharyngeus muscles.

The foregoing presentation is simplified for ready grasp. There are a number of explanatory details that should be added. Ignoring individual muscles and taking the pharynx as a unit, there is a general arrangement of two layers of muscular fibers: the inner layer approximately longitudinal, and the outer layer circular. In swallowing a large bolus, the longitudinal fibers shorten and, being fixed above, pull up the entire pharynx to grasp the bolus,

return it. Mastication and insalivation are not absolutely essential to swallowing, but an unprepared bolus may stick even when there is no muscular paralysis, and a small object like a pill, being no bolus at all, may be difficult to swallow. Liquids, obviously, require no mastication, but liquid foods are better assimilated if they are well insalivated.¹ In case of loss, structural or paralytic, of any part of the mechanism of deglutition the remaining factors vicariously do the best they can to make up the loss, and their vicarious efficiency usually improves with use.

The efferent impulses that activate the muscles concerned in swallowing emanate from the medulla just above the alae cinereae on the

MUSCLES CHIEFLY CONCERNED IN DEGLUTITION AND THEIR NERVE SUPPLY

Muscles

Mylohyoid

Hyoglossus

Styloglossus

Levator veli palatini (includes salpingopalatinus and salpingopharyngeus)

Tensor veli palatini

Glossopalatinus

Pharyngopalatinus

Constrictor pharyngei sup

Constrictor pharyngei med

Constrictor pharyngei inf

Cricopharyngeus*

Esophageal muscular coat (striated and unstriated fibers)

Respiratory muscular system

Innervation

Trigeminal

Hypoglossal

Hypoglossal

Pharyngeal plexus

Otic ganglion (Vth)

Pharyngeal plexus (spinal accessory)

Pharyngeal plexus

Pharyngeal plexus

Pharyngeal plexus and glossopharyngeal

Glossopharyngeal pharyngeal plexus, and inferior laryngeal

Glossopharyngeal pharyngeal plexus and inferior laryngeal

Vagus and sympathetic (Meissner's plexus, Auerbach's plexus)

Vagus, sympathetic laryngeal, and various coordinating

* Anatomists describe the fibers of the cricopharyngeus as part of the inferior constrictor, but its counter action in swallowing shows it to be an independent muscle.²⁻³ It relaxes as the inferior constrictor contracts.

the circular fibers then contract in wavelike succession from above downward, not merely squeezing the bolus but also forcing it in the downward direction. The muscular circle is not complete, there is a "dead" segment anteriorly, formed by the larynx, but this dead segment is carried along in the movements, as anyone can feel in his own neck during swallowing. The hyoid bone goes with the larynx, the thyrohyoid muscle helping to drag the larynx up under the tongue. For greater facility in the pharyngeal grasp and downward squeeze of the bolus, as well as in its later progress, the bolus is prepared by mastication and insalivation. The tongue throws the food out between the upper and lower teeth, the cheeks and lips retain and

floor of the fourth ventricle. The nerve trunks that probably serve as pathways to the muscles are shown in the above table (see also under "Syndromes Associated with Laryngeal Paralysis"). Most of the motor nerves have afferent neurons, and there are doubtless other afferent pathways variable or as yet undetermined.

Etiology and Pathology of Paralysis—The causes of paralysis of one or more of the muscles concerned in deglutition are the same as enumerated in connection with paralysis of the larynx and paralytic syndromes (q.v.). Bulbar troubles, such as acute inferior poliomyelitis, chronic progressive bulbar disease manifested as a labioglossolaryngeal (it is also

pharyngeal) paralysis and myasthenia gravis, are the chief central lesions. In pseudobulbar palsy the manifestations are bulbar but the lesion is above the medulla in the corticobulbar pathways. Paralysis affects movements rather than individual muscles. Spasticity may be noted. Diphtheria and botulism are the commonest of toxic causes. Syphilis is the most frequently causative infective disease. Wounds and tumors near the jugular foramen may be the cause as well as nuclear lesions (see under 'Syndromes Associated with Laryngeal Paralysis' and Fig. 398).

Symptoms—Complete paralysis of the voluntary swallowing muscles, as in bulbar paralysis, is accompanied by complete inability to swallow anything. Some degree of distress, in the epigastrium, behind the sternum, or in the back may be complained of when only the involuntary muscles of deglutition are paralyzed. Stagnant secretion is often to be seen in the pyriform sinuses, denoting lack of aid of swallowing function in esophageal drainage of oral and retronasal secretions. Overflow of the stagnant secretions into the larynx may cause troublesome cough, if the sensory nerve of the larynx, the superior laryngeal, is not also paralyzed. Emaciation and dehydration are conspicuous symptoms especially in cases in which the true significance of the patient's complaint of inability to swallow has been misinterpreted as a manifestation of hysteria. In the advanced stages of progressive bulbar palsy, and in the late stages of pseudobulbar paralysis, the general symptoms of these conditions are added. There are immobility of the face, stupid expression, open mouth, drooling, paralysis of the cheeks, lips, tongue, palate and of muscles of mastication, dysarthria, with or without aphonia, dysphagia, rejection of food through the nose on attempting to swallow, and dyspnea. The whole appearance is that of imbecility, yet the mind is clear. The first symptom in bulbar paralysis is usually in the tongue, the speech is indistinct (dysarthria) and the bolus is difficult to manage in swallowing, though the constrictors are not yet affected.

Diagnosis—In all progressive forms of bulbar trouble the paralysis of the lips and tongue is fully developed and conspicuous before the pharynx is paralyzed. The palate hangs limp as a curtain. The paralysis of constrictors is readily noted by absence of contraction when inspected with the direct laryngoscope, the

cricopharyngeal crescent normally brought into view with this instrument is absent if the cricopharyngeus is paralyzed. The absence of the pinching constriction by the cricopharyngeus muscle, which normally presents the chief difficulty in esophagoscopy, is obvious to any one who has had experience in esophagoscopy. In over 10,000 esophagoscopy studies made without anesthetic the writer did not note absence of the pinching shut of the lumen at the cricopharyngeal level in a single instance unless there was paralysis or disease of the cricopharyngeus. In the cases of pseudobulbar paralysis the lesion is above the medulla in the corticobulbar pathways hence the paralysis is bilateral, and often spastic. Though a patient with bulbar paralysis, myasthenia gravis for example, is unable to swallow anything the ordinary soft rubber stomach tube can be passed without resistance. The diagnosis of the paralysis of the autonomic wave of peristalsis is made by the absence of the wave on fluoroscopic examination made while the patient is swallowing the radiopaque mixture. In some cases the wave is feeble, and the flaccid enlargement of the whole esophagus denotes a condition that has been called *atony of the esophagus*. Fibrosis of the esophagus (*q v*) must be differentiated as else where herein described by Mosher.

Treatment—Treatment depends on the cause, and may be systemic or surgical. One phase, however, pertains to all cases, namely, the prevention of dehydration and dietary deficiencies. Specific, written instructions as to the diet, the amount of fluids that must be taken in twenty-four hours and the times at which they are to be taken must be given to the nurse or to a responsible member of the family. General verbal directions to the patient are certain to result in deficiency. The condition of the patient on arrival is often pitiable, and needlessly so. So long as any power of swallowing remains, soft solids are less difficult to swallow than either solids or liquids.

Prognosis—The prognosis is that of the cause. In cases following diphtheria recovery is the rule. In syphilitic cases, with early diagnosis and prompt energetic treatment, the prognosis is often good. When due to a small hemorrhage, recovery is often fairly good. Progressive bulbar paralysis and myasthenia gravis are hopeless as to function and life. A few patients survive acute inferior poliomyelitis but, if deglutition was involved, tubal feeding is usually re-

quired for the remainder of life Pseudobulbar paralysis is almost hopeless and the duration may be long Acute bulbar conditions, hemorrhage, thrombosis, softening, are not of wholly unfavorable prognosis, and, if the patient survives, the chances of recovery of swallowing function are not bad Life in any case of paralysis of deglutition may depend on the perfection of tubal feeding

Body Origin, Philadelphia, W B Saunders Company, 1936

5 Negus V E The Mechanism of the Larynx St Louis C V Mosby Company 1929

DISORDERS OF DEGLUTITION OTHER THAN PARALYSIS

SENSORY PARALYSIS OF THE ESOPHAGUS

The sensory nerve supply of the esophagus is not accurately determined It has two phases (1) the sensations of contact, thermal changes, and pain, (2) the sensory mechanism necessary to create afferent impulses The complete loss of the latter would mean a break in the reflex arc necessary for swallowing and consequently partial paralysis of peristalsis If any area remained unaffected afferent impulses would still be produced, and such areas usually do remain, though their location is usually not definitely determinable The esophageal mucosa is not delicately sensitive to thermal changes as anyone can determine for himself by swallowing coffee or tea that is uncomfortably hot The discomfort is felt above the level of the cricopharyngeal gateway, but after this is passed no sensation is felt until the hot liquid passes through the hiatal gateway The contact and the pain sense have the same limits of area For this reason paralysis of sensation of the esophagus itself is not noticed subjectively The esophagoscopy taking of a specimen of tissue produces no sensation of pain Injudicious traction on a large tightly impacted denture, for example, may be painful but the pain is from the drag on periesophageal tissues, the pleura especially

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Occasional slight incoordination in the mechanism of swallowing, as described under "The Physiology of Deglutition," occurs within normal limits Everyone has had the experience of a crumb of food or a drop of water intruding in the larynx If, however, this should occur often, suspicion of disorder would justify diagnostic studies

Spasm of the Inferior Constrictor; Alternating Relaxation and Spasm of the Cricopharyngeus Muscle —When the larynx is lifted forward the cricopharyngeal crescent is seen When the esophagoscope is insinuated the orbicular fibers of the upper end of the esophagus join in a tight pinching shut In any action they probably thus join, and they should be considered as acting together The cricopharyngeus muscle normally is always tonically contracted so that the upper esophageal orifice is always shut against inspiration of air when the thorax expands in the respiratory cycle to draw air into the lungs This is demonstrated every time an esophagoscopy is done The pinching shut of the upper end of the esophagus by the cricopharyngeus compels the esophagoscopist to wait As soon as the cricopharyngeus yields to the gentle but steady pressure of the esophagoscope and the distal end enters the thoracic esophagus, the esophagus "breathes" similarly to the trachea, because the cricopharyngeal gate is held open for the passage of air In some patients who complain of a sensation as of "a lump in the throat" there is an alternate relaxation and contraction of the cricopharyngeus muscle The condition, like hiccough, is made worse by fixation of attention upon it In these, as in all cases of esophageal symptoms, the possibility of any symptom being due to beginning organic disease must be kept in mind No negative opinion should be given without an esophagoscopy examination Moreover, the diagnostic esophagoscopy in addition to excluding the possibility of organic disease will usually cure

the alternate relaxation and contraction of the cricopharyngeus muscle

Aerophagia.—The swallowing of air is a normal part of the function of deglutition. As we have pointed out, it greatly assists the swallowing act by prevention of the negative air pressure that would otherwise occur from the descent of the bolus in a closed tube such as the esophagus is when the cricopharyngeus resumes its tonic closure immediately after the bolus has passed this momentarily opened point. Negative pressure would hold the bolus. In some cases, however, the habit of swallowing air alone is acquired, sometimes it is accompanied by belching of the swallowed air in a noisy manner. Horses often acquire the habit (called cribbing) when deprived of bulky diet, as when fed on grain without hay, they do not belch the air but retain it to distend the stomach, which is apparently uncomfortable from the deficiency in bulk. In man the air may be retained in either the stomach or in the esophagus. In the latter it may cause a permanent diffuse dilatation. It probably does no particular harm, but like other habits it can be abolished by will power. Aerophagia and noisy belching are often used as a start in acquiring a pharyngeal voice after laryngectomy.

Retropéristalsis of the Esophagus.—In this movement there is not usually a continuous upward-moving wave demonstrable as exactly like the normal downward moving peristaltic wave, but it is certain that in most cases there is a reverse order in the sequence by the opening, first of the hiatal and, second, of the cricopharyngeal gateway, as in vomiting. There is also an elastic compressive recoil of the esophageal wall, if the esophagus contains enough food, gas, or liquid to fill it. The esophagus contracts best when it is at least slightly distended. If contractions are to be noted in retropéristalsis they are usually less easily seen as compared to the normal. Retropéristalsis is noted in four forms. One form is associated with regurgitation of food that has not reached the stomach. It may be only occasional and not serious, or may be of so serious a degree as to impair nutrition. It may occur in adults or in children, and sometimes it is associated with "pylorospasm" in infants (See article by Harris P. Mosher entitled "Fibrosis of the Esophagus.") If there is no organic disease found retropéristalsis will disappear after the passage of an esophagoscope of full size for the age of the patient. Repetition

of the treatment may be required. In another form there is only slight occasional epigastric discomfort that lasts only a short time. Esophagoscopy shows no organic disease and the retropéristalsis can be seen in the fluoroscope by using a radiopaque mixture. It requires no treatment. Another form, quite common, is the return of acid gastric contents up the esophagus a variable distance, sometimes only a few centimeters, sometimes reaching the pharynx. It is commonly called "heartburn." Discomfort may be relieved by the ingestion of 15 to 30 grains (1 or 2 gm.) of sodium bicarbonate in water, but the condition of the stomach should be studied. The fourth form is the ordinary belching that requires no treatment. It cannot be too strongly emphasized that, in all the foregoing conditions, the diagnosis should not be made until after organic disease has been excluded by esophagoscopy.

Psychosomatic Dysphagia.—Swallowing is a peculiar function in that, though done voluntarily, it is best done inattentively. Even an average normal person may have trouble swallowing a pill with water if the attention is fixed beforehand on the pill and on the swallowing of it. Sometimes, if the pill lodges, as pills thus swallowed are prone to do, this will be the start of the maladjustment complex with the phases of escape mechanism that are often erroneously called "hysteria." In other cases there is a psychic trauma from shock. In our records are cases in which this form of dysphagia started from fright caused by having swallowed substances suspected of being injurious—lye in one case, an insecticide in another, antiseptic tablets instead of emulsion in two cases. In some of our cases the psychic trauma was the accidental swallowing of lye, not by the patient but by a relative of the patient. Cases due to supposed swallowing of insects or small animals, such as frogs, have been reported. In psychosomatic dysphagia the chief symptom may be that the patient refuses to attempt to swallow food, or he may refuse only certain kinds of food. In either case there may be secondary symptoms of avitaminosis or other deficiency. Rarely the patient may be obsessed with the idea of inability to swallow water, and this may be to the extent that dehydration may develop. Almost invariably there are other symptoms indicating the associated maladjustment. A tentative diagnosis may be made on the basis of the unstable temperament, while studying the case, but this

should never be taken as conclusive. To do so would risk repeating the old error of diagnosing globus hystericus, on the basis of complaint of a feeling as of a "lump in the throat," in case of cancer or other organic lesion. Organic stenosis by such conditions as web, cicatrix, tumor, ulcer, fibrosis, or paralysis should be ruled out by esophagoscopy before making a diagnosis of psychosomatic dysphagia. Malingering must be excluded. The malingerer is not psychopathic. He knows perfectly well he can swallow, but pretends he cannot do so, the pretense being to escape duty or perpetrate fraud. In the treatment of psychosomatic dysphagia it is of primary importance to guard against the patient being told by anyone, even an unaware practitioner, that there is nothing the matter with him, and that he can swallow if he wants to. Such a statement is often made and it enormously increases the difficulty of cure. The patient should be taken seriously, but should not be told his condition is serious. If the esophagoscopy has revealed no organic disease he should be frankly told so and it should be added that he will get well and will be able to swallow perfectly. If his confidence in his ability to swallow does not immediately enable him to do so, his confidence must be obtained and he must be helped and encouraged to overcome his maladjustment, as is regularly done by neurologists (see "Psychosomatic Aphonia"). The condition is curable in practically all cases, with perfect restoration of the swallowing function. Recurrences may call for repetition of the treatment.

Achlorhydric Anemia with Dysphagia.—A clinical type of functional difficulty in swallowing has been reported by Plummer and Vinson and is known as their syndrome.² Synonyms are *glossitis with dysphagia*, and *anemic dysphagia*. The affliction usually occurs in edentulous women over forty years of age and is characterized by achlorhydria, splenomegaly, secondary anemia, fear of choking, glossitis, fissures at the corners of the mouth, weakness, and shortness of breath. The diagnosis is made by these features after cancer has been eliminated from the diagnosis by esophagoscopy. Studies of the blood will exclude pernicious anemia. Treatment is already begun when the diagnostic esophagoscopy is done and no serious disease is found. The patient is reassured and is convinced by the swallowing of solids. Further demonstration of the freedom of ob-

struction may be made with the stomach tube or mercury bougie. Iron is given for the anemia. The splenomegaly disappears. Almost all patients recover. There is often a thin web present in these cases, and this is why esophagoscopic dilatation is so effective.

Obstruction at the Upper End of the Esophagus.—The most frequent organic obstruction in this region is cancer (*q v*). Next in frequency is web and other forms of fibrosis described by Mosher (see page 687).

Obstruction at the Lower End of the Esophagus.—The most common obstructive disease in this region is fibrosis secondary to infection as shown by Mosher (see page 687). Next in frequency is cancer (*q v*). Any form of obstruction in the lower end of the esophagus may result in accumulation of food in the thoracic esophagus and the accumulation causes more or less dilatation, this occurs even in conjunction with cancer, though the superjacent dilatation associated with this malady is not as extensive as that in obstructive diseases of longer duration. If there is time enough the dilatation may reach huge proportions. This degree of dilatation is possible only because it is slowly attained over a period of years. Rapid distention of normal wall is promptly fatal from rupture as in connection with the treatment of segmental cicatricial stricture. Diffuse dilatation is sometimes caused by long delay in opening of the hiatal closing mechanism. This condition called "achalasia" is treated by dilatation with Mosher's pneumatic dilator or Hurst's mercury bougie or both. Symptomatic relief is obtained but the associated diffuse dilatation is permanent. Peptic ulcer may occur from acid juice brought from the stomach by retroperistalsis, or derived from acid secreted in the cells in the thoracic part of the stomach in cases of gastric hernia (see "Hiatal Hernia"). Varices occur in cases of Banti's disease and in association with other maladies. They are obstructive when large. The veins of this region are a part of the anastomosis between the portal system and the vena cava. Any obstruction to the portal or caval circulation naturally causes back pressure and varices are enormous. Thinning of the walls may cause hematemesis or fatal hemorrhage. Diagnosis of varices is by esophagoscopy, carefully and gently done, as it always should be. Treatment for esophageal hemorrhage is by bismuth subnitrate given dry on the tongue, 60 grains (4 gm) every hour for,

say, 3 doses, less often thereafter Rest in bed is essential and transfusion may be needed when bleeding is copious, morphine is contraindicated because of its tendency to cause vomiting The presence of blood in the stomach may do this anyway and thus the hemorrhage is increased Esophagoscopy injection of a sclerosing solution, such as sodium morrhuate, as advocated by Moersch⁶ is of at least palliative value General therapeutics should be directed toward lessening portal and caval venous pressure The etiology, pathology, and treatment of obstruction of the lower end of the esophagus are included in the section on "Fibrosis of the Esophagus" by Harris P Mosher, who has made a special study of this region

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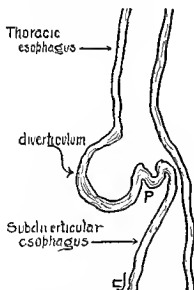


Fig 533—Schema and esophagoscopy view illustrating a case of pulsion diverticulum of the thoracic esophagus due to cicatrices (at P) which also caused stenosis The diverticulum produced no symptoms but the stenosis did Esophagoscopy dilatation cured the stenosis causing the symptoms to disappear, but the diverticulum could be demonstrated esophagoscopically and by roentgenogram to have remained unchanged The patient was still free from subjective symptoms two years later

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DIVERTICULUM OF THE ESOPHAGUS

A pouch leading off from the esophagus is known as a diverticulum of the esophagus The term is often misapplied to pouches leading off from the hypopharynx (qv)¹

Etiology.—The esophagus is a long, loose, redundant, resilient bag Normally it empties



Esophagoscopy view

itself quickly Any obstruction that delays emptying will make dilating pressure on the resilient walls above the obstruction There is no limit to the extent of dilatation except the extent of available surrounding space, provided the dilatational process is slow enough, usually years are required If the walls are uniform in strength throughout, the dilatation will be uniform When normally supported by the cricopharyngeus and the hiatus the dilatation will be evenly spindle shaped or fusiform Any increased strength, as an adhesion or a scar in the walls, or in the surrounding structures, will be

resistant to dilatation. Conversely, any weak point in the wall or the structures that support the wall will yield more readily than will the normal wall, normally supported, and the yielding is pouchlike in form. The weak point in the wall may be congenital and is probably atavistic. An actual pouch at birth is exceedingly rare but a congenital weak point is common. In other words, *congenital diverticulum* is oftener potential than developed. The usual division into *pulsion*, *traction*, and *congenital* is inaccurate as two or all of these factors enter more

weak point in the periesophageal supporting structures. In the earlier stages of development the walls of the sac show but slight alteration of the cellular structure. Well developed sacs may show in their walls the effect of pathologic changes of stretching, chronic infection, and fibrosis (q 1.)

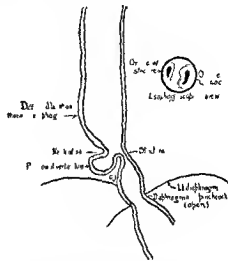


Fig. 535—Schematic illustration of a pulsion diverticulum of the thoracic esophagus that developed in the course of two years above a cicatricial stricture due to peptic ulcer. Diagnosis made and development observed at intervals by esophagoscopy. Symptomatic cure followed esophagoscopy treatment.

Symptoms—The chief symptoms are discomfort felt back of the sternum, or in the epigastrium, or in the back, or in two or all three of these locations. Gentle filling of the mouth with food on lying down is usually noted in case of a large sac, it is not accompanied, usually, with retching or nausea.

Diagnosis—Diagnosis is made by esophagoscopy and roentgen ray examination. The appearances are shown in Figures 533, 534, and 535. The mucosa in the interior of the sac is whitish, furled, and pasty and usually contains particles or masses of food unless thoroughly washed out by the use of a stomach tube before endoscopy.

Treatment—Treatment is palliative, unlike pharyngeal diverticulum the symptoms are, usually, mild—too mild to justify thoracotomy. The sac should be washed out with a glass of water taken after each meal and followed by a short period of pillowless recumbency. If the patient finds the glass of water to be an excess of fluid, no fluid need be taken during the meal.



Fig. 534—Diverticulum of the lower thoracic esophagus in a woman aged sixty-two. Regurgitation of food when recumbent was an annoying symptom. The full regular pouch and narrow neck found at esophagoscopy suggested congenital rather than cicatricial origin. A mere dimple at birth, the diverticulum enlarged as the result of pulsion.

or less into the causation of all diverticula of the esophagus as well as of the hypopharynx. A causative factor in some cases is fibrosis (q 1.)

Pathology—The mucosa may herniate through a weak point in the muscular coat or both mucosa and wall may herniate through a

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GASTROSCOPY

The examination of the esophagus is completed when in an adult the esophagnoscope of 9 mm lumen and 53 cm length enters the stomach and explores the folds of the collapsed stomach within about 5 cm radius in each direction. Diagnostic exploration beyond this

stomach are thin, clear, and watery, they gush into the gastroscope as soon as the hiatus is passed. The sound is the signal that the gate is opened. Later they gush in from time to time as folds are moved aside. The quantity is not great and is quickly drawn away automatically through the drainage canal of the gastroscope, the inlet of which is always kept at the bottom of the visual field by keeping the handle uppermost. In the search for a lesion and for the taking of a specimen of tissue the stomach is explored by progressive traverse. That is after exploring down to the greater curvature, the tube mouth is moved laterally about 2 cm, and the withdrawing travel explores a new field. Then a lateral movement affords a fresh field during the next insertive travel. This is repeated until the entire explorable area has been covered. If desired to push the folds away from the

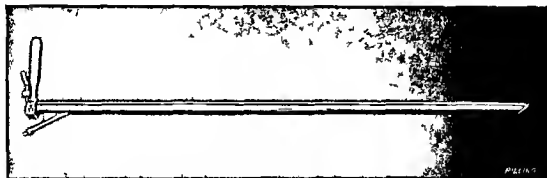


Fig. 536—Open tube gastroscope for removal of foreign bodies from the stomach and for taking specimens of tissue for biopsy. A window plug may be used when it is desired to inflate the stomach.

area is usually done with the lens system flexible gastroscope of Wolf Schindler.¹ The optical system, however, interferes with the insertion of instruments so that the open tube gastroscope is required for the removal of specimens of tissue or of gastric foreign bodies. The open tube gastroscope (Fig. 536) may be closed with a window plug having a rubber diaphragm with a central perforation for forceps when it is desired to inflate the stomach. The rigid open tube gastroscope can be introduced into the stomach of any patient whose mouth can be opened and whose spine is normal. The technic is the same as given for esophagoscopy. If the stomach is distended a 9 mm by 60-cm gastroscope may be necessary, but for most purposes in almost all adult patients the 9 mm by 53 cm is sufficient. A baby requires a 5 mm by 30-cm tube, an older child a 7 mm by 45 cm size. The secretions in the normal empty

tube mouth it is best done with a hand ball from a hand ball atomizer while momentarily occluding the proximal tube mouth with the thumb. The positive pressure tube of the electrical aspirator is dangerous for this purpose, it inflates the stomach too rapidly. Abdominal breathing is helpful because when the open tube gastroscope is admitting air freely to the stomach each respiratory expansion limited to the abdominal walls draws air into the stomach just like a bellows is filled. To get the full benefit of abdominal inspiration, however, the patient must be trained beforehand to breathe abdominally at command. While passing through the thoracic esophagus thoracic breathing is more helpful than abdominal. Gastroscopy for foreign body is considered in the section on Foreign Bodies in the Air and Food Passages.

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BIPLANE FLUOROSCOPY AS AN AID IN BRONCHOSCOPY, ESOPHAGOSCOPY, AND GASTROSCOPY^{1 2}

Indications.—In considering the indications for the use of biplane fluoroscopic guidance in bronchoscopy, we think, of course, of *foreign*

very often patient, careful work for an hour or more, or even a second bronchoscopy, may be required before success is attained. Biplane fluoroscopic guidance is of value in the removal of screws and nails lodged head upward, since fluoroscopic guidance enables us to determine whether or not our forceps blades open widely enough, and whether or not we are allowing them to open too widely. Any large or sharp-pointed object is handled more satisfactorily if the guidance of the biplane fluoroscope is used. Foreign bodies in the stomach can be removed only with fluoroscopic guidance.

The introduction of lipiodol for *bronchography in cases of bronchial stenosis* is best accomplished by fluoroscopically-guided bronchoscopy (Fig 538), though for routine bronchography we prefer one of the indirect catheter methods, using the fluoroscope as an aid in the accurate placement of the catheter, as described elsewhere in this book.

Though "costophrenic bronchoscopy" is generally thought of chiefly as a method of removing foreign bodies from the periphery of

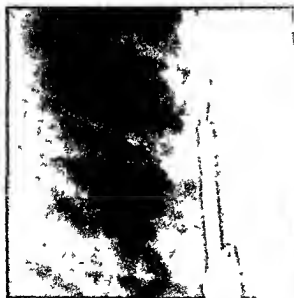


Fig 537—Films taken on the biplane fluoroscopic table showing a common pin about to be withdrawn through the costophrenic bronchoscope.³ The films are placed in such a way as to show relations as they are when the bronchoscopist is looking at the fluoroscopic screen—to the left the vertical ray view, and to the right the horizontal ray view.

bodies in the periphery of the lung,³ especially common pins to be removed by "costophrenic" bronchoscopy (Fig 537). It must be borne in mind that while the guidance of the biplane fluoroscope may make an otherwise impossible removal of a foreign object by bronchoscopy possible, it does not always make it easy, and

the lung the exploration of peripheral bronchi in order to find the one leading to an area of density in the outer lung field with a view to biopsy or the drainage of an abscess cavity (Fig 539) is also "costophrenic bronchoscopy," and may be done to advantage under the guidance of the biplane fluoroscope.

Another important use the endoscopist makes of the biplane fluoroscope is to guide him in

sequent treatment by either peroral or retrograde bougienage



Fig 538 —Bronchogram made by fluoroscopically guided bronchoscopic bronchography. Because of the high degree of obstruction produced by a tumor of the right main bronchus it was impossible to fill the right upper lobe with lipiodol except by introducing a flexible tipped metal tube through a bronchoscope. Film to the right was taken on the fluoroscopic table and is placed upside down to simulate the view obtained by the bronchoscopist

the procedure of *combined peroral and gas trostomic esophagoscopy* for "threading of the esophagus" in cases of tight stricture or total atresia. This procedure is always a hazardous

Apparatus—When Dr. Edward Chamberlain first became associated with Temple University, about ten years ago, the importance of biplane fluoroscopic guidance in bronchoscopy



A



B

Fig 539 —Films taken on the biplane fluoroscopic table to record the relations of a certain branch bronchus to a cavity in a tuberculous patient with artificial pneumothorax. Note that the anteroposterior view (A) shows the tip of the opaque bougie not quite entering the cavity, though the lateral view (B) suggests that it has entered

one, but, if the greatest care is exercised, fluoroscopic guidance will increase the number of these cases in which a thread may be successfully introduced for use as an aid in sub-

was discussed with him. He was told what had been the chief shortcomings of the various types of apparatus used previously, and he set to work to design a new fluoroscope which would

overcome them. For example, it had always been difficult for the head holder to support the patient's head in the proper position and yet not be crowded by the horizontal ray tube or its supports. Therefore, Chamberlain substituted a tube at a distance of four feet, behind an aluminum panel, with the added good effect that distortion was reduced with no loss of

paratus the tube for the vertical ray is sunk under the floor, also a distance of four feet from the patient. A "transom latch" mechanism is used to hold the screen in the horizontal position when used with the vertical ray. Separate foot switches mounted upon a common base control the horizontal and vertical ray units.



Fig. 540—The Chamberlain biplane fluoroscope. The tube for the horizontal ray is back of the aluminum panel A. The tube for the vertical ray is beneath the floor under the bakelite panel B. Both tubes and the fluorescent screen C are carried on an integral supporting frame. By this arrangement both the horizontal ray and the vertical ray are always centered on the fluorescent screen wherever the screen may be placed.

visibility, but rather increased perception of detail. As an additional improvement, both tubes were mounted on a frame of steel tubing which traveled on rails and on this framework also the single screen used alternately in the two planes was mounted with the result that it moved at all times exactly with the tubes. At first the original old tube under the table was used for the vertical ray, but in the present ap-

An important feature of the Chamberlain apparatus (Fig. 540) is the pair of automatic self starting electric clocks which record the exact duration of exposure of the patient's skin to the vertical and horizontal rays respectively (Fig. 541). These two roentgen ray beams having been previously calibrated by means of the ionization chamber, the total permissible exposure time for either beam is known in ad-

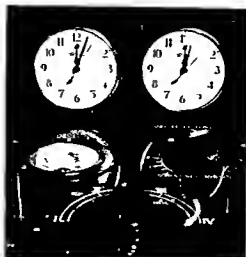


Fig 541—Part of the control panel of the biplane fluoroscope, showing the automatic electric clocks for timing the exposures. No matter how many times the exposure is interrupted, or changed from one beam of rays to the other, the clocks indicate the total exposure time for each beam. At the beginning of each biplane fluoroscopic procedure the hands are turned to twelve o'clock. When the above photograph was made the horizontal ray had been in operation for a total of three minutes and seven seconds (see clock at left). Accurate ionization-chamber calibrations of both ray beams enables the radiologist to determine accurately, in advance, the maximum safe time limit for each beam.



A



B

Fig 542—A, The biplane fluoroscope during horizontal-ray fluoroscopy. When the radiologist moves the screen cephalad, caudad, ventrad, or dorsad in relation to the patient, this movement is rigidly transmitted to the tube. Thus the beam of roentgen rays is at all times centered on the screen. The radiologist's left foot depresses the horizontal ray foot switch, which energizes the horizontal ray tube and also the horizontal ray time clock. B, The biplane fluoroscope during vertical ray fluoroscopy. The radiologist has turned the screen upon its hinges and his left foot has shifted to the "vertical-ray foot switch" which energizes the vertical ray tube and the vertical ray time clock.

vance, and the possibility of overexposure or limitation of the procedure are thus both eliminated

The author feels that the Chamberlain fluoro scope herein described and more fully described in a joint article with Chamberlain¹ possesses certain distinct advantages for bronchoscopic work (1) complete protection of patient and attendants against the possibility of electric shock (2) absence of any interference with the work of the bronchoscopist or his assistants (3) maximum protection of patient radiologist, and others present against exposure to roentgen rays (4) central rays of both beams geometrically set at 90 degrees apart, truly vertical and truly horizontal, respectively, (5) minimum of enlargement and distortion of the fluoroscopic images (6) ease of manipu-

lation and centering, and instantaneous change-over from vertical ray to horizontal and vice versa (Fig 542)

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PART VII. FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

FOREIGN BODIES IN THE UPPER AIR AND FOOD PASSAGES

A foreign body is substance not normally connected with its surroundings. In the air and food passages it may be *endogenous*, originating within the body, a sequestrum for example, or *exogenous*, having entered from without.¹

Foreign Body in the Nasal Cavity.—Foreign bodies frequently enter the nasal cavities through the anterior nares, occasionally they enter through the posterior nares in vomiting or in cases of lost or paralyzed velum palati. If small they are expelled by sneezing or escape in secretions. If of a size or shape to lodge they may not be expelled. The annoyance leads an adult to seek help, but in a child tolerance is soon established and the incident is forgotten. In a number of cases an unsuspected foreign body in the nose, also in the nasopharynx, has been discovered in our clinic as the result of the rule that all children should have a roentgen ray examination including the nose and abdomen as well as neck and chest.¹ Dried beans and maize swell and become impacted, these are usually uncooked and they may undergo the first stage of germination. Beads and buttons may become encrusted with salts. There is rhinorrhea at first. *Purulent rhinitis* follows. Usually it is unilateral. The possibility of foreign body should be excluded in every case of suppurative or bloody discharge from the nose, even if the patient has tuberculosis, syphilis, or other disease causative of purulent rhinitis. Even in cases of sinus disease, foreign body may be present in the nose. In case the foreign body is not demonstrated by roentgen ray study, careful search is necessary. Good illumination, shrinkage of the mucosa and the granulations, if any, with cocaine and adrenalin solution, and careful inspection will reveal almost any foreign body in the nasal chambers. An alligator forceps is most often useful in removal. Open safety-pins, staples, tacks,

pins, and the like call for the same general principles of solution of mechanical problems as when they lodge in the lower air and food passages even though the traumatic risks of ruthless tearing out of the foreign body are not so great. Insects, maggots and other larvae, and larger protozoa should be removed with forceps and the nasal chambers washed with a copious spray of sodium bichlorate solution. As a rule, solutions to kill the maggots are undesirable as a dead maggot involves a septic risk, especially when entrenched in the *maxillary* or *sphenoidal sinuses* or ethmoidal cells. In these regions flushing in the usual way is sufficient. Penetrating projectiles are considered under "*Injuries of Paranasal Sinuses in Warfare*."

Endogenous Foreign Body in the Nose.—Sequestra of cartilage and bone occur from syphilis and from traumatic or operative fragmentation. *Rhinolith rhinitis* is the name given to the reaction, usually unilateral, due to a rhinolith. This endogenous foreign body is composed of mineral salts, chiefly of calcium and magnesium, encrusted upon an organic or inorganic nucleus. Foreign-body accumulations of coagulated or desiccated inflammatory products have given rise to names compounded with the words "*coryza*" and "*rhinitis*," though many of them cannot be considered morbid entities. Thus recorded are *coryza caseosa*, *rhinitis caseosa*, *cholesteatomatous rhinitis*, *rhinitis foetida*, and *pseudomembranous, membranous, fibrinous, croupous* and *diphtheritic rhinitis*. There are two forms of diphtheritic rhinitis, the well-known acute toxic disease, and a peculiar chronic form in which the organism, *Corynebacterium diphtheriae*, is present in large numbers but without extension and without toxemia or other systemic symptoms. In the latter, in the patients we have seen, there has been no evidence of communicability. Differential diagnosis as among the various kinds of endogenous foreign bodies just mentioned is made through microscopic

and bacteriologic studies of removed material Treatment is carried out by forceps removal followed by cleansing with a copious spraying of a saturated solution of borie acid in warm water Prognosis is good except in the rare cases of cerebral complications

Foreign Body in the Nasopharynx—In some instances a foreign body has reached the nasopharynx from the food passages during emesis, or digital efforts at removal, or otherwise Rarely it may be pushed back through the nose by a child or psychoneurotic person The symptoms are subjective sensation of contact, pain, blood-stained secretions, or, later, pus, usually fetid Diagnosis is by roentgen ray and examination with the small mirror Removal is by small forceps with upward curve Laryngeal forceps may be used through a Yankauer nasopharyngeal speculum Care should be taken to prevent loss downward into the lower air passages or esophagus, by placing the patient in recumbent posture with low head ¹

Foreign Body in the Tonsil or Pharynx—It is quite common for fish bones or spicules of other bones to lodge in a crypt of the tonsil Toothbrush bristles and pieces of toothpick do the same thing If not found by direct inspection with a tongue depressor a small mirror should be used to inspect the posterior surface of large tonsils, the supratonsillar and infratonsillar fossae, the posterior surface of the velum, the pharynx, and the larynx If no foreign body is seen the posterior surface of the tonsils, if hypertrophic, should be carefully palpated with the index finger, the point of a foreign body, like a spicule of bone, may be felt projecting little if any above the orifice of a crypt If a foreign body is found it can be removed under good illumination with direct laryngoscopic forceps and tongue depressor If nothing is found roentgen ray examination should never be neglected There may be a pin or similar object lodged somewhere If nothing is found and the patient still has the sensation of some thing in his throat every time he swallows he should be given bismuth subnitrate 15 grains—(1 gm) dry on the tongue every third hour and he should be asked to return the following day for re-examination if the sensation of foreign body persists A hasty conclusion that a sensation of foreign body is imaginary may be a cause for regret ¹

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FOREIGN BODIES IN THE DEEPER AIR AND FOOD PASSAGES

This subject requires separate consideration because special methods of diagnosis and treatment developed within the last forty odd years have reduced the former appalling mortality in this kind of case to less than 2 per cent, and thereby, moreover, has been created a new department of medical science.¹

Lodgment of a foreign body in the deeper air and food passages is a common occurrence The serial number of such cases at our clinic at the time of writing is 4259

Etiology—There has been only one etiologic study based upon a great mass of material ¹ It is therein clearly shown by the analysis of the histories of 3112 cases that the causes are classifiable into six groups (1) Personal factors, such as age (peanuts—99 per cent occur in children), sex, occupations (work or play), social condition (poverty) and place of residence. (2) Failure of the patient's normal protective mechanism, including sleep, alcoholic incoordination, epileptic seizure, unconsciousness (3) Physical factors, expression of emotions, activities, posture (4) Dental, medical, and surgical factors (5) Properties of the foreign body itself (6) Carelessness, this was found in the form of (a) putting inedible substances in the mouth, (b) preparation of food, (c) hasty eating and drinking, (d) permitting children to play while eating, (e) giving peanut candy to children who have not yet erupted their molars ¹

Pathologic Changes Due to Foreign Body in the Bronchi—In the bronchi the character of the foreign body has an important bearing on the pathologic tissue changes it causes Some substances cause little or no reaction unless by reason of size or shape they cause bronchial obstruction (q v) High-carat gold is an example Other metals are less irritating than vegetal substances, iron and steel by ionization inhibit bacterial activity, but oxidation pro-

duces traumatizing physical qualities of roughness of surface and sharpness of edge, especially the edge of scales. All vegetal substances irritate the tracheobronchial mucosa, the reaction is diffuse and its severity quickly results in obstructive swelling. Peanuts, usually roasted, are so oily they do not swell and the angularity permits partial passage, whereas beans and maize swell enormously, and are not irregular, thus cooperating with the mucosal swelling in forming a tight corking of the invaded bronchus.¹ The irritating qualities of vegetal substances are in some instances chemical, in others allergic.¹ Natural rubber objects exert the same reaction as other vegetal substances. Synthetic rubber is a physical, not a chemical, imitation, its reactionary effects, doubtless variable, must remain for future determination.

The pathologic changes in tissue may be summarized as follows: (1) A smooth, metallic nonobstructive foreign body will produce, at most, only a slight local congestion of vessels in the part of the mucosa in contact with the intruder, so long as it remains smooth and nonobstructive. (2) A metallic foreign body that is rough and obstructive, or if and when it acquires these qualities by corrosion, causes localized inflammation and mucosal swelling that perfects the occlusion of the bronchus, a stop valve type of obstruction (*q v*). A one-way valve type of obstruction may have preceded complete occlusion. Shortly after occlusion of a bronchus, the tributary area, segment, lobe, or entire lung, collapses, thus means atelectasis without pneumothorax. If atelectasis persists for many weeks infective mucosal inflammation begins in the atelectatic area, because of the lowered resistance resulting from absence of ventilation and drainage. The infective agents are the pyogenic bacteria of which there are a few in the bronchi, too few and too weak to overcome the resistance of the mucosa so long as good ventilation and drainage are maintained. Pus accumulates and a condition of drowned lung is established, that is to say, the natural passages are filled with pus. Abscess formation, with breaking down of the walls of the natural passages, does not develop for many weeks, often months, of sojourn of this kind of foreign body. The period is even longer in case of a smooth, noncorrosible, impervious foreign body. There is a high degree of resistance to septic invasion of the lung through the barrier of the bronchial mucosa. (3) In case of a vegetal

foreign body, there is a violent reaction in the mucosa, a *vegetal bronchitis*, starting within a few hours in a baby, within a day or two in a two year old child. In case of a foreign body like a fragment of peanut kernel, which has facets that prevent a perfect corking, there is, for a short time, a space for by-passage of air. This by-pass produces, with the normal respiratory enlargement and diminution of lumen, a valvular action that results in atelectasis from pumping air out of the tributary area, or emphysema from pumping air into it (Figs 544, 546). Soon, however, within a few days in babies, a week or two in older children, the swelling of the mucosa incidental to the violent reaction closes the by passage, residual air in the tributary area is absorbed, obstructive atelectasis is quickly followed by drowned lung. Whereas this condition of complete obstruction may not be reached for months in case of a metallic foreign body in an adult, it may be reached in a few weeks in case of a child under two years of age, in an even shorter period in an infant a few months old. In contrast with the more or less angular fragment of peanut kernel, a tightly-fitting bean of rounded contour quickly corks the bronchus. For a short time the bean may lift to let air out on expiration, being sucked back at the beginning of inspiration, thus producing atelectasis of the tributary area rapidly by pumping air out. Swelling of the mucosa combined with swelling of the bean soon puts a stop to this one-way valvular action. If any residual air should remain after the out pumping action it is quickly absorbed. With the bean the atelectatic and drowned lung sequence is usually the most rapid as compared to any other foreign body. These patients, if the bean is not removed, never survive long enough to go through the lengthy period, often years, of slowly progressing supuration so characteristic of cases of overlooked metallic foreign body. In the latter class of cases, granulations may form in two weeks, rarely sooner, often not for a month or two. The foreign body remains in its bed of granulations indefinitely. The granulations are exuberant, flabby, and in course of months they build up more and more fibrotic tissue which later contracts and forms a stricture at the proximal end of the foreign body. This stricture in time may attain obliteration of lumen, though usually a small fistulous lumen remains and discharges pus at each compression of the tribu-

tary segment by the tussive squeeze.² All of the foregoing statements are based on clinical facts in hundreds of cases at our bronchoscopic clinic, most of which have been published.¹ Parenthetically, it may be mentioned that these observations, originally made on foreign body, have clarified the conception of pulmonary pathologic conditions in the living and have corrected the misinterpretation of physical signs formerly based on autopsic findings.^{1, 3}

Bronchoscopic Appearances of Foreign Bodies.—If seen within a few days of its invasion, the presenting part of a foreign body may resemble its prior appearance. Nut kernels and maize are soon coated with pus, of which they appear to be composed. With the exception of high carat gold and a few alloys of the nonferrous metals, all foreign bodies are usually darkened with oxides or sulfides. This discoloration is augmented or modified by adherent secretions often blood stained. In the cases of vegetal bronchitis there is a constantly recurring flood of secretions to be aspirated. In other cases there is usually little secretion early, but later there may be much secretion and, after granulations form, it will be blood stained, this will paint the picture opaque pink. In cases of drowned lung great quantities of pus will well up with each tussive squeeze.³

Endogenous Foreign Bodies in the Lungs and Bronchi.—In acute tracheobronchial and pulmonary diseases the commonest endogenous foreign bodies are clots and plugs of agglutinated exudates, membranes, and crusts. These often cause atelectasis of pulmonary segments by occlusion of segmental bronchi. The obstruction is often of the one-way valvular mechanism that pumps the air out causing segmental atelectasis (*q v*) usually mistaken for pneumonic consolidation. The obstruction may be of the stop valve type in which air is absorbed by the circulation, or of the one way ingress type causing obstructive emphysema. All of these types of bronchial obstruction are seen every day at a busy bronchoscopic clinic. In the chronic cases of pulmonary disease, sequestra, broken down nodes, and sloughs occur as endogenous foreign bodies. *Broncholiths* are less frequently encountered. Broncholithiasis is the name given a condition in which a broncholith is coughed up or is removed bronchoscopically. We have removed a number at the Bronchoscopic Clinic at Temple University Hospital.¹ They were encountered in the course of bronchoscopic

examination or treatment of disease of the bronchi or lungs. They are secondary as pathologic products, though until removed they cause or perpetuate chronic bronchial suppuration. They derive from three sources. *Pneumoliths* are cast off calcified caseated masses that have become free and have found their way into a bronchus by ulcerative processes. Broncholiths are formed in a bronchus by the deposit of salts of lime and magnesium on the stagnant pathologic exudates accumulated in the course of chronic bronchial disease, usually bronchiectasis. Chemically both pneumoliths and broncholiths are found to be composed of phosphates, sulfates, and carbonates of lime and magnesium, with, in some instances, traces of oxides of iron or other metals. These two kinds of lung stone are endogenous foreign bodies. The third kind, the *siliceous broncholiths*, are probably of exogenous origin. We have removed these siliceous foreign bodies in the form of small particles that were angular under the microscope and as small cylindrical lung stones that had the appearance of having been formed in small bronchi. Anthracotic material including some granules of silica was found in a number of cases in which anthracotic peribronchial nodes had ulcerated through into the bronchial lumen.^{1, 4} Calcareous and phosphatic material was found incrusting on foreign bodies of long sojourn in the bronchi.⁴ The diagnosis as to presence of broncholiths can sometimes be made with the roentgen ray, but usually broncholiths are not sufficiently radiopaque to show through the shadows of pathologic tissue with which they are surrounded. They call for removal because they are foreign bodies and as such perpetuate suppuration. They can always be bronchoscopically removed when they occupy a bronchus and the pulmonary prognosis is improved thereby. If in pulmonary tissue, they can be removed if radiopaque by fluoroscopic bronchoscopy (*q v*), but removal is seldom if ever advisable.

Various pathologic processes produce *bronchial casts* as endogenous foreign bodies. The most perfect branching specimens encountered in the course of our bronchoscopic work were in cases of fibrinous bronchitis. In diphtheria the material is shorter and more friable. Fragmentary casts we have seen in cases of typhoid fever, measles, pneumonia, and tuberculosis. The large casts should be removed, if encountered, lest they cause asphyxia. The fragmentary

casts usually come away with aspirated or expectorated secretions.¹

Pathologic Changes Due to Foreign Body in the Esophagus—Changes in the wall are slight and slow in developing unless the foreign body is particularly traumatizing. As a rule, foreign bodies are adjusted by the esophageal yielding movements to a position least likely to cause trauma, for example, safety pins turn over and go down with spring end leading, the point trailing. If any kind of foreign body becomes impacted there will be, after a long time, months usually, an erosion of the epithelium and ulceration with a building up of fibrous tissue around the foreign body. Spontaneous perforation sometimes occurs. If it is sudden, subcutaneous and mediastinal emphysema may follow, but the esophageal wall, though very thin, is yielding, which is its main defense from trauma. If a foreign body ulcerates through the esophageal wall slowly, the surrounding tissue layers are sealed together in advance by inflammatory processes, this usually inhibits extension of sepsis. Eventually, however, extension will occur though the foreign body seldom follows the suppurative progress. In some cases in which a foreign body in the esophagus had been overlooked for years the suppuration that it caused had reached the left bronchus, then the pleural cavity, and had established a fistula discharging externally, yet the foreign body remained embedded in the esophagus. Liquid food, however, often came out of the fistula.¹

Esophagoscopic Appearances of Foreign Bodies—During the first few days there may be little change in appearance of either foreign body or mucosa. After a few days most metallic foreign bodies turn dark from formation of oxides or sulfides on the surface. In the cervical esophagus most of the foreign body will be hidden by normal looking folds of esophageal wall, in the thoracic esophagus negative pressure at each inspiration will open the lumen of the esophagus and expose the edge of the central zone of the foreign body to view. In long sojourn cases a foreign body in the thoracic esophagus may be hidden by folds of inflammatory mucosa and there may be granulations, rarely stricture. In such cases the esophagus should be kept under monthly observation for a year, so that any tendency to stricture can be cured by esophagoscopic bougienage.¹

Symptoms and Diagnosis of Foreign Body in the Air and Food Passages—From the accumu-

lated data concerning over 3000 cases at our clinic the following summary of symptomatology and diagnosis was compiled.¹

Initial symptoms are choking, gagging, coughing, and wheezing, often followed by a symptomless interval. The foreign body may be in the larynx, trachea, bronchi, nasal chambers, nasopharynx, fauces, tonsil, pharynx, hypopharynx, esophagus, stomach, intestinal canal, or may have been passed by bowel, coughed out or spat out, with or without the knowledge of the patient. Initial choking, or other manifestation, may have escaped notice, or may have been forgotten. When a child has been known to choke, gag, and cough while suspected of having something in his mouth the case should be regarded as one of foreign body until proved otherwise by every diagnostic means we possess. Cyanosis is not uncommon, and asphyxia may terminate the case in the initial stage. Pain or other subjective sense of presence of foreign body is not felt in the bronchi, rarely in the trachea. It is felt in the esophagus at each swallowing act. Children under two years of age, however, cannot tell us of their sensations.¹

Laryngeal Foreign Body—One or more of the following laryngeal symptoms may be present: hoarseness, croupy cough, aphonia, odynophagia, hemoptysis, wheezing, dyspnea, cyanosis, apnea, subjective sensation of foreign body. Croupiness in foreign body cases, as in diphtheria, usually means subglottic swelling. Obstructive foreign body may be quickly fatal by laryngeal impaction on inspiration, or on abortive hecic expulsion. Lodgment of a non-obstructive foreign body may be followed by a symptomless interval. Direct laryngoscopy for diagnosis is indicated in every child having laryngeal diphtheria without faucial membrane (No anesthetic, general or local, is needed.) In the presence of laryngeal symptoms, think of the following: (1) A foreign body in the larynx (2) A foreign body loose or fixed in the trachea (3) Digital efforts at removal (4) Instrumentation (5) Overflow of food into the larynx from esophageal obstruction due to the foreign body (6) Esophagotracheal or esophagobronchial fistula from ulceration set up by a foreign body in the esophagus, followed by the leakage of food into the air passages (7) Laryngeal symptoms may persist from the trauma of a foreign body that has passed on into the deeper air or food passages or that has been coughed or spat out (8) Laryngeal symptoms (hoarseness,

croupiness, etc.) may be due to digital or instrumental efforts at the removal of a foreign body that never was present. Laryngeal symptoms may be due to acute or chronic laryngitis, diphtheria, pertussis, infective laryngotracheitis, and many other diseases (10). Deductive decisions are dangerous (11). If the roentgen-ray examination reveals no abnormality, laryngoscopy (direct in children, indirect in adults) without anesthesia, general or local, is the only means of making a laryngeal diagnosis (12). Before doing a diagnostic laryngoscopy, preparation should be made for taking a swab specimen and for bronchoscopy and esophagoscopy.¹

Tracheal Foreign Body—In case of tracheal foreign body the (1) audible slap, (2) palpatory thud, and (3) asthmatic wheeze are pathognomonic. The tracheal flutter has been observed in a patient in whose trachea a watermelon seed had lodged. Cough, hoarseness, dyspnea, and cyanosis are often present. Diagnosis is by roentgen ray, auscultation, palpation, and bronchoscopy. One should listen long for the audible slap, best heard at open mouth during cough. The asthmatic wheeze is heard with the ear at the patient's open mouth. It resembles the sound of asthmatic wheezing but is unrelated to asthma. A history of initial choking, gagging, and wheezing is important if elicited, but is valueless negatively.¹

Bronchial Foreign Body—Initial symptoms are coughing, choking, asthmatic wheeze, et cetera, as mentioned. There may be a history of these or tooth extraction. At once, or after a symptomless interval, cough, blood streaked sputum, metallic taste, or special odor of foreign body may be noted. Nonobstructive metallic foreign bodies afford few symptoms and few signs for weeks or months. Obstructive foreign bodies cause atelectasis, drowned lung, and eventually pulmonary abscess. Lobar pneumonia is an exceedingly rare sequela. Vegetable organic foreign bodies, such as peanut kernels, beans, and watermelon seeds, cause at once violent laryngotracheobronchitis, with toxemia, cough, and irregular fever.¹ Bones, animal shells and inorganic bodies after months or years produce changes which cause chills, fever, sweats, emaciation, clubbed fingers, incurved nails, cough, foul expectoration, hemoptysis, in fact, all the symptoms of chronic pulmonary sepsis, abscess, and bronchiectasis. These symptoms and some of the physical signs may

suggest pulmonary tuberculosis, but the apices are normal and bacilli are absent from the sputum. Every acute or chronic chest case calls for the exclusion of foreign body.

The *physical signs* vary with conditions present in different cases and at different times in the same case.¹ Secretions, normal and pathologic, may shift from one location to another, the foreign body may change its position, admitting more, less, or no air, or it may shift to a new location in the same lung or even in the other lung. A recently inspired pin may produce no signs at all. The signs of diagnostic importance are chiefly those of partial or complete bronchial obstruction, though a nonobstructive foreign body, a pin for instance, may cause limited expansion or, rarely, a peculiar r le or a peculiar auscultatory sound. To interpret the signs it is necessary to remember that there are four kinds of obstruction,^{1 3 5} namely, (1) bypass valve obstruction, like a partially closed valve, (2) check-valve obstruction in which air passes in but not out (emphysema), (3) check-valve obstruction in which the air emerges but cannot enter, and (4) completely shut valve in which the air cannot pass either in or out, the retained air becoming absorbed (atelectasis). It must be remembered also that while these four types of obstruction are usually seen separately and distinctly, they may alternate from shifting of the foreign body or secretions, may be present simultaneously in different areas, or may follow each other in the same lobe or lung.^{1 5} The most nearly characteristic physical signs are (1) limited expansion, (2) decreased vocal fremitus, (3) impaired percussion note, and (4) diminished intensity of the breath sounds distal to the foreign body. Complete obstruction of a bronchus followed by drowned lung adds absence of vocal resonance and vocal fremitus, thus often leading to an erroneous diagnosis of empyema. Varying grades of tympany are obtained over areas of obstructive or compensatory emphysema. With complete obstruction there may be tympany from the collapsed lung for a time. R les in a case of complete obstruction are usually most intense on the uninvaded side. In partial obstruction they are most often found on the invaded side distal to the foreign body, especially posteriorly, and are most intense at the site corresponding to that of the foreign body. A foreign body at the bifurcation of the trachea may give signs in both lungs. Early in a foreign-body case, dimin-

ished expansion of one side, with dullness, may suggest pneumonia in the affected side, but absence of, or decreased, vocal resonance, and absence of typical tubular breathing should soon exclude this diagnosis ¹

always include all the structures from the nasopharynx to the tuberosities of the ischia, other wise a foreign body may be overlooked, or if one is found others may be overlooked in case of multiple foreign bodies (Figs 546-547). Ex-



Fig. 543—Obstructive emphysema due to check valve (one way valve) action caused by a radiopaque foreign body. The heart and mediastinum are displaced to the uninvaded (left) side during expiration because air trapped in the invaded (right) lung prevents it from deflating. The left (uninvaded) lung empties itself of so much air on expiration as to give a misleading shadow suggesting pathologic change though it is normal. The sidewise movement of the heart at each respiratory cycle is the essential diagnostic point distinguishing obstructive emphysema from obstructive atelectasis (cf. Figs 544-545).

The roentgen ray is the most valuable diagnostic means but careful notation of physical

pert ray work will show not only all metallic foreign bodies and many of less density, such as teeth, bones, shells, and buttons, but also will demonstrate the presence of nonradiopaque foreign bodies by the evidences of the bronchial obstruction they produce. If no evidence of foreign substance is found, a diagnostic bronchoscopy should be done in all cases of unexplained obstruction. Peanut kernels and all other vegetal substances for a few days at least produce obstructive emphysema of the invaded side. Fluoroscopy shows the diaphragm flattened, depressed, and of less excursion on the invaded side, at the end of expiration, the heart and the mediastinal wall move over toward the uninvaded side and the invaded lung becomes less dense than the uninvaded lung, from the trapping of the air by the expiratory, valve like effect of obliteration of the "forceps spaces" that during inspiration afford air ingress between the foreign body and the swollen bronchial wall. This partial obstruction causes *obstructive emphysema*, which must be distinguished from *compensatory emphysema*, in which the ballooning is in the unobstructed lung because its fellow is wholly out of function through complete "corking" of the main bronchus of the invaded side (Figs 543, 544). *Obstructive atelectasis* occurs when the obstruction is complete, air below the obstruction

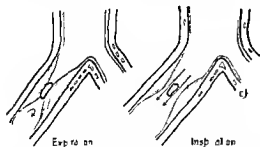


Fig. 544—Mechanism of obstructive emphysema of the right lung caused by a peanut kernel in the bronchus. On expiration the foreign body is embedded in swollen mucosa preventing exit of air. On inspiration the enlargement of the diameter of the bronchus opens a small by passage for ingress of air as indicated by the darts. Immediately upon the start of the expiratory phase the normal expiratory diminution of bronchial diameter closes the small by passage, trapping the air below. Repetition of the process twenty odd times per minute soon "pumps" the tributary lung full of air an obstructive emphysema. Though this illustration is schematic it is not theoretic. The mechanism is a clinical fact first observed bronchoscopically and now seen daily in a busy bronchoscopic clinic in cases of foreign body, endogenous or exogenous, as well as in various diseases of the lung. It is an expansile one way valve type of obstruction.

signs by an expert should be made in all cases, preferably unbiased by knowledge of roentgen-ray findings. Roentgen ray examination should

is absorbed, the lung collapses, and the mediastinum moves over to satisfy the vacuum. This occurs often within twenty-four hours in cases of beans and maize, which quickly swell to a tight fit. These two roentgen-ray signs—obstructive emphysema and obstructive atelectasis (Fig 545)—are diagnostic, but to understand them the four kinds of bronchial obstruction above mentioned must be realized, and to get the evidence on a film two exposures must be made, one at full inspiration and one at the end of complete expiration, for comparison.¹

Diagnosis of site of lodgment by physical signs and roentgen ray depends upon accurate knowledge of the location of segments of lung and their orifices (see under "Applied Anatomy of the Tracheobronchial Tree from the Endos-

through from the esophagus into the trachea or bronchus, or (3) trauma inflicted on the larynx during attempts at removal, digital or instrumental, the foreign body still being present or not. Pre-existing esophageal disease, especially cancer in case of adults,¹ must always be thought of when anything is arrested in the esophagus.

Diagnosis is by the roentgen ray, first without, then, if necessary, with a capsule filled with an opaque mixture. Flat objects, like coins, always lie with their greatest diameter in the coronal plane of the body, when in the esophagus, in the sagittal plane, when in the trachea or larynx. Lateral, anteroposterior, and sometimes also quartering roentgenograms are necessary. One taken laterally, low down on the neck but clear of the shoulder, will often show a



Fig 545—Obstructive atelectasis due to a bean in the right main bronchus of a child four years old. The heart and mediastinal structures are displaced to the invaded (right) side and they remain displaced during both inspiration and expiration. This is the essential point of differential diagnosis distinguishing obstructive atelectasis from obstructive emphysema. In emphysema the heart and mediastinal structures move back to place on inspiration (cf Fig 543).

copist's Viewpoint") For many years we have used the insufflation of bismuth subcarbonate to assist in this localization.⁶

Esophageal Foreign Body.—After initial choking and gagging, or without these, there may be a subjective sense of a foreign body, constant or, more often, on swallowing. Odynophagia and dysphagia, aphagia, or drooling may or may not be present. Pain, substernal or extending to the back, is sometimes present. Hematemesis and fever may occur from the foreign body or from rough instrumentation. A child may hold his head stiffly to one side, or may show a preference for a prone position. Symptoms referable to the air passages may be present due to (1) overflow of the secretions on attempts to swallow through the obstructed esophagus, (2) erosion of the foreign body

bone or other semi-opaque object invisible in the anteroposterior exposure. The most frequent site of lodgment is in the cervical esophagus, just below the cricopharyngeal constriction.¹ The chief reason for this lodgment is the weakness of the peristaltic musculature, which is sufficient to carry on downward a bolus of well-masticated and properly insalivated food that the powerful constrictors have forced past the relaxed cricopharyngeus, but not sufficient to carry down the physically different foreign body.

Treatment of Foreign Bodies in the Deeper Air and Food Passages.—In a general way it may be stated that if the foreign body is in the larynx it should be removed by direct laryngoscopy, if in the tracheobronchial tree or lung, by peroral bronchoscopy, if in the esophagus,

by peroral esophagoscopy No other methods are worthy of a moment's consideration Waiting for spontaneous expulsion is dangerous Blind methods of dealing with esophageal foreign body are extremely dangerous Groping in either tracheobronchial tree or esophagus under the fluoroscope is rarely successful and is at

the contrary, are readily removed by fluoroscopic gastroscopy (*q v*) The question as to the necessity for removal is for decision in the particular case¹ The majority of foreign bodies that reach the stomach unassisted go on through natural passages provided the natural protective means are not impaired by the administra-



Fig. 546 —An example of the importance of having a roentgen ray examination include the entire trunk After esophagoscopy removal of the bur of safety pins from the cervical esophagus the child a one year old baby was detained in the hospital in order that the needle might be watched It perforated and only prompt laparotomy saved the baby's life

tended with high mortality Foreign bodies in the stomach may be removed by peroral gastroscopy, if indicated *Intestinal foreign bodies* should be watched daily and removed by external operation if they lodge in one location for four or five days If allowed to remain longer in one place they are likely to perforate by ulceration *Foreign bodies in the stomach on*

tion of cathartics or changes in diet. This however does not mean that esophageal foreign bodies should be pushed into the stomach by blind methods which are always dangerous, or even by esophagoscopy, because a swallowed object lodged in the esophagus can practically always be removed through the mouth In cases of nontraumatizing foreign body in the stomach

it may be wise, if the pylorus is of normal size, to wait a month or two under daily watch. If by roentgen-ray studies it is determined that the presence of a pyloric stenosis will prevent the passage of the foreign body, even though not potentially traumatizing, it should be removed by peroral gastroscopy.¹ Pins of various types and similar objects have passed safely through the intestines; but the occasional case of ulcerative perforation renders gastroscopic removal advisable, if the patient is seen before the foreign body has left the stomach. Certain objects reaching the stomach may be judged too large to pass the normal pylorus. These should be removed by gastroscopy when such decision is made.¹ The most dangerous foreign body to be

be stated that if unremoved, foreign bodies in the trachea, bronchi, and esophagus ultimately prove fatal. About 2 to 4 per cent of bronchial foreign bodies are coughed up. About 99 per cent of foreign bodies in the lung can be bronchoscopically removed through the mouth. After bronchoscopic removal of a foreign body in a bronchus the patient gets well in about 98 per cent of the cases, even after the foreign body has set up extensive pathologic changes from prolonged sojourn.¹ This is in marked contrast to pulmonary suppuration of other cause.¹ Foreign bodies in the esophagus can be removed by esophagoscopy through the mouth in almost all cases; it has never been necessary in our experience of over 1500 cases to remove



Fig. 547.—Another example of the importance of requiring that roentgen-ray examination include all regions from the nasopharynx to the pelvis. The pin that was supposed to have been swallowed would have been missed by routine roentgen-ray examination. It was invisible at routine examination of the fauces.

allowed to pass through the pylorus is one that is too long to pass the turns of the duodenum.¹ This is an unfavorable location for removal by abdominal section and such foreign bodies can be safely removed from the stomach through the mouth by gastroscopy. As a criterion it may be stated that a foreign body 2 inches long, or longer, is likely to lodge at the turn of the duodenum in a child two years old. A large safety pin will lodge at the same point, and a small one may stick anywhere in trying to turn over; a medium size that cannot turn over has the best chance of going through, usually spring end first. About 25 per cent of safety pins lodge.¹ Some ferrous foreign bodies can be removed from the stomach with an alnico magnet.

Prognosis of Foreign Body in the Deeper Air and Food Passages.—In a general way it may

a foreign body by external esophagotomy except in a single instance, in which there was stenosis due to pulsive and tractive impaction.¹ The public press reports show that foreign body is the cause of many deaths by asphyxia before the victim can reach a physician. A large piece of candy may asphyxiate a child; a small bit in a bronchus may dissolve.

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MECHANICAL PROBLEMS OF FOREIGN BODY EXTRACTION

To avoid the pitfalls and disasters such as beset much of the early work in the endoscopic removals of foreign bodies from the air and food passages it must be remembered that before any traction is made on any foreign body it must be definitely known just what traction will cause that particular foreign body to do. For example, in case of an open safety pin lodged point upward, traction would almost invariably cause perforation and death, whereas proper solution of the mechanical problem will result in almost 100 per cent of successful removals.¹

Preliminary Study—The first step in the solution of the mechanical problems is the study of the roentgenograms made in at least three planes (1) anteroposterior, (2) lateral, and (3) the plane corresponding to the greatest plane of the foreign body. Having ascertained the size and shape and character of the foreign body the experience tables of our clinic¹ are got out and similar cases are studied, noting the age of the patient, size of tube, size of foreign body, kind of forceps, the point of seizure, and the other details of the respective methods. We call these tables 'canned experience'. Over 3000 cases have been tabulated.¹ From them the bronchoscopist can learn the difficulties encountered in our clinic in cases similar to the one confronting him and how the difficulties were handled. We constantly make such use of these tables.

The next step is to put a duplicate of the foreign body into the open manikin board previously referred to, and try to simulate the probable position shown by the roentgenograms, so as to get an idea of the bronchoscopic appearance of the probable presentation. As shifting may change the presentation the duplicate foreign body is turned into as many different positions as possible, so as to educate the eye to assist in the comprehension of the largest possible number of presentations that may be encountered at the bronchoscopy on the particular patient. For each of these presentations a method of disimpaction, disengagement, disentanglement, or version and seizure is worked out, according to the kind of foreign body.

Selection and Use of Forceps for the Particular Case—It cannot be too strongly stated that preliminary selection and test of forceps with a duplicate of the foreign body should be made in every case. It has often been a sad sight to see a child arrive at our clinic very ill or moribund from prolonged efforts, elsewhere, to remove a foreign body with forceps the utter uselessness of which could have been determined in a moment by testing on a duplicate. It is necessary, in some cases, to have different forceps ready for different presentations. Forceps jaws must expand sufficiently to take in the foreign body, if not in every diameter at least in the diameter of the selected presentation and portion to be grasped.

Prepared by this practice and the radiographic study, the endoscopist introduces the bronchoscope into the patient. The location of the foreign body is approached slowly and carefully to avoid overriding or displacement. A study of the presentation is as necessary for the bronchoscopist as for the obstetrician. It should be made with a view to determining the following points: (1) The relation of the presenting part to the surrounding tissues. (2) The probable position of the unseen portion, as determined by the appearance of the presenting part taken in connection with the knowledge obtained by the previous roentgen ray study, and by inspection of the films upside down on the view box in sight of the bronchoscopist. (3) The version or other manipulation necessary to convert an unfavorable into a favorable presentation for grasping and disengagement.

The standard forceps (Fig 541) have a powerful grasp and are used on dense foreign bodies which require considerable pressure on the

object to prevent the forceps from slipping off. All forceps should be tested for grasp to see if they will slip off when traction is made. For more delicate manipulation, and particularly for friable foreign bodies, the lighter forceps

third, or ring finger, in the other ring. These fingers are used to open and close the forceps, while all traction and pulsion are to be made by the right index finger, which has its position on the forceps handle near the stylet, as shown in

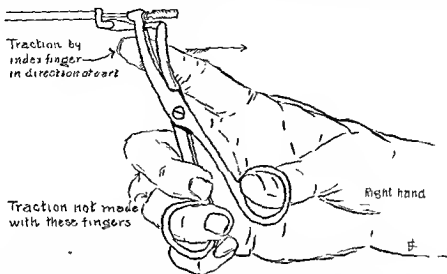


Fig. 548.—Proper hold of forceps. The right thumb and third fingers are inserted into the rings while the right index finger has its place high on the handle. All traction is made with the index finger, the ring fingers being used only to open and close the forceps. If any pushing is deemed safe it may be done by placing the index finger back of the thumb-nut on the stylet.¹

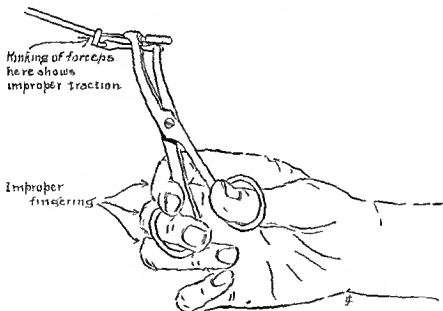


Fig. 549.—Improper use of forceps. Forceps can be so constructed as to do away with the springing upward here shown; but the delicacy of touch essential to safe and efficient work, as illustrated in Figure 548, would be thus destroyed.¹

are best. Spring-opposed handles on a pair of forceps render any delicacy of touch impossible. Stuff expanding-spring jaws are necessary in certain cases. Forceps are to be held in the right hand, the thumb in one ring, and the

Figures 548 and 549. The impulse to seize the object as soon as it is discovered must be strongly resisted. A careful study of its size, shape, position, and relation to surrounding structures must be made before any attempt at

extraction The most favorable point and position for grasping having been obtained the closed forceps are inserted through the bronchoscope, the light reflex is noted as the forceps pass the distal lamp and emerge from the distal end The forceps are then advanced until they lightly touch the foreign body, they are then allowed to expand and are further advanced far enough to get a secure grasp on the intruder If there are no potentially traumatizing points on the foreign body, it is held until the tube mouth is advanced against it If it is too large to come out through the tube, it is held against the tube mouth, the grasp of the forceps being firmly

one When searching for such objects as pins and needles especial care must be taken not to override them Pins are almost always found point upward, and the dictum can therefore be made, "*Search not for the pin, but for the point of the pin*" If the point be found free, it should be worked into the lumen of the bronchoscope by manipulation with forceps and the lip of the tube It may be then seized with the forceps and withdrawn Should the pin be grasped by the shaft, it is almost certain to turn crosswise of the tube mouth, where one pull may cause the point to perforate, enormously increasing the difficulties by transfixation, and perhaps resulting

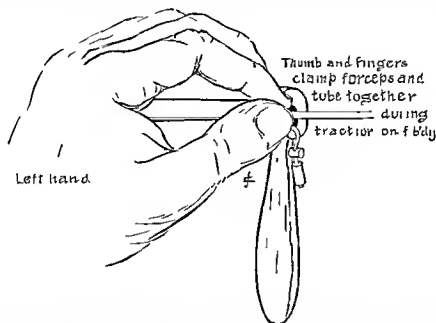


Fig. 550—Method of anchoring the foreign body against the tube mouth. After the object has been drawn firmly against the lip of the endoscopic tube the left finger and thumb grasp the forceps cannula and lock it against the ocular end of the tube, the other fingers of the left hand encircle the tube. Withdrawal is then done with the left hand, the fingers of the right hand maintaining firm closure of the forceps.¹

maintained by the fingers of the right hand, while all traction for withdrawal is made with the left hand, which firmly clamps forceps and bronchoscope as one piece. Thus the three units are brought out as one, the bronchoscope keeping the cords apart until the foreign body has entered the glottis (Fig. 550). Coins require presentation and a flatwise grasp. They should be kept close against the tube mouth and turned sagittally before reaching the glottis. If in the esophagus, they should be rotated to the frontal plane. Pins, needles, and similar long pointed objects are in two groups: (1) bendable pins, and (2) breakable pins and needles. It is often desirable to bend a pin, undesirable to break

fatally.¹ Pins in the costophrenic angle, or elsewhere in the periphery of the lung, require costophrenic bronchoscopy under fluoroscopic guidance.¹ The sheathing and protective methods for pins apply also to the removal of tacks. Double-pointed tacks and staples require the special staple bronchoscope and forceps.¹ Hollow metallic objects are best held with one of the forceps jaws inside and one outside. Hard smooth-surfaced globular objects like ball-bearing balls are best held with the ball forceps. The same is true of globular-shaped glass beads. Oval-shaped beads are securely held with Gordon's bead forceps. They require extremely gentle and careful use to avoid dangerous

trauma The forceps should not be allowed to expand until they have touched the bead. If gentle traction meets with resistance the bead must be released and a fresh grasp made. Safety pins in a bronchus have the point not far distant from the keeper. The method of dealing with them is given in a subsequent paragraph.

Foreign bodies in the esophagus are usually lodged in the cervical portion just below the cricopharyngeus muscle. The powerful inferior constrictor by its swallowing movement forces them into the upper end of the esophagus where there is nothing but peristalsis to send them on

placement of forceps.¹ The rotation forceps (Fig. 541) are most frequently serviceable for bones, dentures, and other large impacted foreign bodies, because they hold well yet allow the foreign body to swing. Spicules of bones are manipulated like a needle or a pin to seek and sheath the point before any traction is made. Transfixed bones are seized by one end with rotation forceps so they will swing to bring their long axis parallel to the long axis of the esophagus. In case of a large foreign body tightly im-

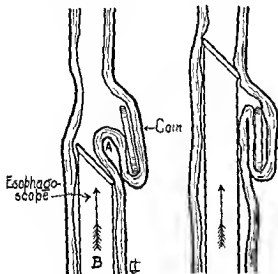


Fig. 551—Illustrating the hiding of a coin by the folding downward of the cricopharyngeal fold. The muscular contraction throws the beak of the esophagoscope upward while the interposed tissue prevents the tactile appreciation of contact of the foreign body with the side of the tube after the tip has passed over and beyond the foreign body. Other folds may in rare instances act similarly in hiding a foreign body from view. This overriding of a foreign body is likely to cause dangerous dyspnea by compression of the party wall. Not infrequently a foreign body is overridden because it remains anterior to the beak of the esophagoscope.¹

downward, and this peristalsis is relatively weak as applied to a relatively large foreign body in this region. The foreign body thus located is hidden in folds because of atmospheric pressure on the neck. In this position it is likely to be overridden by the esophagoscope (Fig. 551). When a foreign body reaches the thoracic esophagus the negative intrathoracic pressure on inspiration draws air into the esophagus and this opens up wide "forceps spaces" around the foreign body, greatly facilitating proper presentation at the distal tube-mouth and proper

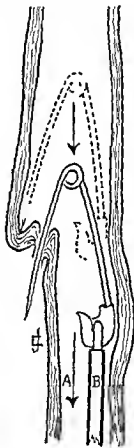


Fig. 552—Schematic drawing of what will happen if the dictum, "advancing points perforate, trailing points do not," is ignored.

placed in the esophagus of an adult, deep relaxing general anesthesia will help to release it. Ether is safest for this. Sodium amyl and other anesthetics that cause muscular contraction increase the impaction. On the other hand, general anesthesia in case of a large foreign body such as a jack, for example, in the esophagus of a child may be rapidly fatal by asphyxia because of the compressibility of the tracheoesophageal party-wall.¹ The best forceps for a jack is the ball forceps applied to one of the four globular extremities. Any presentation

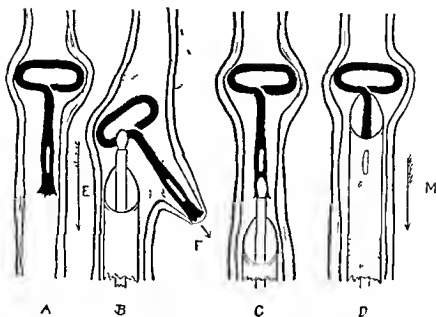


Fig 553 —Mechanical problem of a can opening key lodged in the cervical esophagus. The key was lodged with the sharp, jagged end uppermost as shown in a roentgenogram of the patient and as indicated schematically at A. At B is shown how traction (dart E) on a malpresenting part has forced the key toward a position of transfixion with perforation (F) of the esophageal wall (subcutaneous emphysema was present on admission). At C is shown a proper presentation and seizure at the proper point so that by pushing down the esophagoscope as shown at D the sharp, jagged point of the key is safely sheathed in the esophagoscope for unresisting withdrawal in the direction of the dart M. This illustrates not merely the features of this case but the fundamental principles of endoscopy for foreign body.

other than a globular extremity is to be regarded as a malpresentation that should be converted into a favorable one. *Safety pins in the esophagus* are dangerous (Fig 552). They may be dealt with by sixteen different methods.¹ The most frequently used are (1) *endogastric version*, (2) *endogastric straightening*, (3) the point sheathed and (4) closing methods. In the bronchi the last two methods are the most serviceable. The best form of closer is Clerf's.

Complications and Pitfalls of Esophagoscopy for Foreign Body—Most of these complications are due to one of two things, namely, (1) impingement or insinuation of the esophagoscope (qv) or (2) traction on the presenting part of a foreign body without first determining what may be the result of such traction (Figs 552, 553). The esophagus is exceedingly intolerant of trauma, much more so than any other viscus. If there is slight nonperforating trauma of the esophagus, bismuth (15 grains—1 gm) should be given dry on the tongue every half hour for ten doses, then every three hours thereafter for a few days. This forms an antiseptic coating by its quality of adhesion to the traumatized spot.

If perforation of the esophageal wall has occurred as shown by brawny, tender swelling of the neck, frequent roentgenograms should be made to detect periesophageal abscess at the earliest stage. If this complication should occur, immediate external operation above the clavicle on the side indicated by the roentgenogram is urgently indicated. The approach in this operation is the same as that for pharyngeal diverticulum as described on a previous page.

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PART VIII. GENERAL CONSIDERATIONS

ENDOSCOPIC PHOTOGRAPHY IN OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

The simplest conception of endoscopic photography in otolaryngology is the substitution of a camera for the examiner's eye. However, the camera is infinitely less flexible optically than the eye. If the consequent limitations are ignored, passable photographic results will be obtained by the use of simple apparatus, however, by the use of more complicated specially-constructed apparatus, these limitations may be largely overcome to produce exceptional photographic results. Each of these types of photography serves a particular purpose: the former for simple case-recording, the latter for research and teaching.

There are certain fundamental principles upon which photographic equipment and results may be judged. In the design of any equipment the safety of its use in regard to the patient must be considered paramount. The equipment may be judged by its ability to expose the field completely and cover it with adequate, proper light. It must permit good visualization of the field during finding and, if possible, during filming. The accuracy and depth of focus, the simplicity of operation, and the ease of manipulation are further criteria for judging equipment. Improvements in results are usually obtained through a sacrifice of simplicity in operation and ease of manipulation. Flexibility, or the attempt to design one piece of apparatus to be equally effective in a number of different fields, might erroneously be considered a favorable attribute of certain types of equipment. However, a camera which is a Jack-of-all-trades is disappointingly the master of none. Photographic results may be judged by the accuracy of exposure (color) and the depth of focus, as well as by the composition, *i.e.*, size of field and reference to landmarks, and the relation of the size of the image to the size of the frame.

General Considerations.—Endoscopes—All endoscopes fall into two general classifications—

lens-system endoscopes, such as nasopharyngoscopes and cystoscopes, and open-tube endoscopes, such as bronchoscopes and esophagoscopes. In either case, they should be as large as possible when used for photography, preferably considerably larger than those used in routine examinations. This additional size permits more light to reach field, lens, and film, allowing a greater depth of focus or a shorter exposure time, and it also produces a greater field, including more landmarks. Fields devoid of landmarks are worthless. The rapid scanning of an area which the otolaryngologist does automatically for orientation must be duplicated in motion pictures, or substituted in still pictures by an increase in the size of the field.

The inner surface of the open endoscopic tube becomes an exceptionally high-reflecting surface with cleaning and sterilization. Since such reflections are confusing in the projected picture the inside of the tube may be threaded to break up the reflections and darkened by one of various processes. However, a considerable increase in light value on the field is obtained if the inner surface of the tube is highly polished or nickel-plated. The insertion of a proper mask immediately in front of the film eliminates the photography of the reflections on the inner surface of the tube. The appearance of the projected picture is greatly enhanced by masking these photographed reflections, as well as by masking the portions of the field which are out of focus and confusing.

Illumination—All sources of light must, of course, have the correct color temperature for the particular film being used. Three types of illumination are available: proximal, distal, and a combined proximal and distal. Distal illumination is used with most of the lens-system endoscopes. When used with open tubes, multiple small lamps are placed around the circumference of the endoscope. The advantages are simplicity of construction and ease of manipulation. However, numerous small lamps produce multiple highlights, and, when placed close to the object being photographed, produce marked variations in the intensity of il-

illumination of the field with only slight variations of the distance of the endoscope from the field. Proximal illumination permits the use of lamps of much greater intensity and uniformity and gives the axial lighting similar to that obtained with the head mirror. Combined proximal and distal lighting may be used to increase light values but becomes cumbersome.

Finding and Focusing—Because of the short distance between the lens and the object in otolaryngologic photography as well as on account of the small size of the field, direct finding and focusing are indispensable. Further, more sufficient illumination for these steps preliminary to the actual photography is likewise essential. Direct focusers inserted into magazine cameras are highly satisfactory since the field

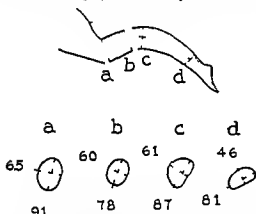


Fig. 554.—Configuration and dimensions (in millimeters) of the external auditory canal. The optical and lighting systems of apparatus suitable for photography of the drum membrane are limited by these measurements. (From Bezold and Sebenmann, *Textbook of Otolaryngology*, translated by J. Holinger, Chicago: E. H. Colegrove Company.)

is viewed through the lens and all parallax is eliminated. The image is projected upright onto a ground glass and is magnified ten times; thus accurate centering and focusing are possible. However, the focuser must be removed before the film magazine may be inserted into the camera for the actual photography, and the movement associated with this operation is liable to change the position of the endoscope. Reflex finders consisting of small 45 degree mirrors placed behind the lens and immediately in front of the film throw the image onto a ground glass viewer and are more satisfactory but they too are removed during the actual filming. A more efficient though more complicated system for finding and focusing consists of a telescope built into the endoscopic system.

This permits constant vision of the field not only for finding and focusing but also during filming. The value of seeing the field while it is being photographed cannot be overestimated.

Fogging—Rhinoscopic, laryngoscopic and bronchoscopic photography is complicated by the condensation of moisture on the lens as the patient breathes and by the gross soiling of the lens if the patient coughs. The use of antifogging preparations advocated for eye glasses has not proved successful. If the preparation is applied in quantity sufficient to prevent fogging it streaks and distorts the image. Therefore the lens must be heated in a manner similar to that used for a laryngeal mirror. An electric heating pad regulated to its lowest temperature is most satisfactory; higher temperatures injure the lens by melting the cement between the glass elements. This means of preventing fogging still necessitates complete cleaning of the lens should the patient cough. A removable heated glass slide inserted in front of the lens is more efficient in that it may be quickly changed should the patient cough during photography. Similarly, when a lighting system is used which depends in part on the reflection of the inner surface of the endoscope, it is desirable to heat the endoscope before use to prevent the condensation of moisture on its inner surface.

Technic—Some understanding of the fundamentals of photography is essential if endoscopic photography is to be attempted. Further, the otolaryngologist himself must be the photographer since he is adept at exposing the field and recognizing the lesion to be photographed. Finally, no matter how simple and foolproof or how complex and elaborate a given piece of photographic equipment may be, practice in the technic of its use is essential in order to obtain the desired results.

For all but otoscopic photography anesthesia is generally necessary in order to have the patient relaxed enough to permit proper finding and focusing. Topical anesthesia is usually sufficient. Endoscopic photography during the administration of ether or any other explosive anesthetic is dangerous because the temperature of the lamps rises above the flash point of those anesthetics and because sparks may occur in the electrical circuits of the apparatus.

The Ear—Photography of the external ear requires no special equipment. The position and size of the drum membrane, however, present

unusual photographic problems. The diameter of the drum membrane averages 9.2 mm. by 8.5 mm., while the 35-mm. long, somewhat spiral auditory canal through which the picture must be taken is, in one place, only 8.1 mm. by 4.6 mm.; further, the drum membrane does not lie perpendicular to the canal, but at an angle of not less than 140 degrees, with the upper edge more proximal than the lower.¹ The problem is to photograph an oblique elliptical

of the canal place significant limitations on the types of illumination and the lenses which may be used in equipment designed to photograph this area.

Several types of cameras are in use which give satisfactory still pictures of the drum head. One of these consists of an extremely small lens system which may be introduced into the canal.² The light source is directed onto the ends of a quartz tube which surrounds the lens

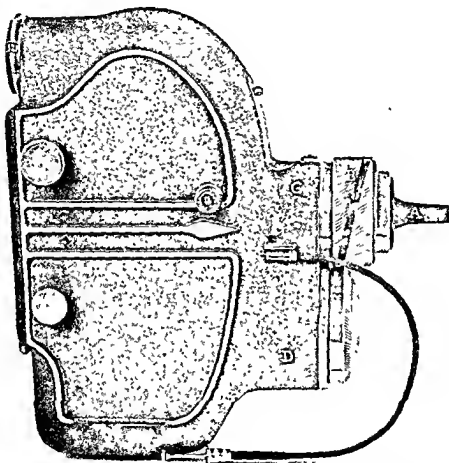


Fig. 555.—Cameron Flash Camera with special focusing mount and ear speculum used for still pictures of the drum membrane. (A) Ear speculum; (B) focusing mount; (C) chamber for lens, mirror, and focusing light; (D) housing for flash bulb and filters; (E) ground glass viewer for reflex focuser. (From Hantman, Irvin: *Arch. Otolaryng.* 34.)

surface through a canal whose smallest diameter is considerably smaller than the ellipse itself (Fig. 554). The importance of these anatomical measurements is twofold: the obliquity of the drum head makes it necessary to have optical and lighting systems which will permit a relatively great depth of focus, approximately 8 mm., if all parts of the drum are to be visualized clearly on one photographic image; the changing diameters and the length

system, thus carrying the light directly into the canal. A second solution of the problem has been suggested by Hantman,³ using a Cameron camera with a special adapter for focusing (Fig. 555). In this camera the light of a flash bulb mounted within the camera is thrown onto a 45-degree mirror, and projected forward into the cavity to be photographed. The lens is placed in the center of the mirror with the focusing adapter holding the ear speculum

mounted in front of it. Focusing is done by means of a reflex finder and a ground-glass viewer. According to Hantman, an image about the same size as the drum head is obtained. Adaptation of the laryngeal flash camera, described by Brubaker and Holinger,⁴ to the field of otoscopic photography provides an additional camera system for this problem. Increased visualization for focusing and greater depth of focus are the advantages of this system.

The use of motion pictures to record the normal and pathologic ear drum has certain advantages over the still camera in spite of the fact that the drum is a relatively stationary object. Motion pictures permit a moving inspection of the drum membrane, simulating the usual otoscopic examination. Photographically, this permits the use of a smaller single field which does not require a great depth of focus. Borrowing a laryngeal and bronchoscopic camera,⁴ such motion pictures have been made. This equipment is described in detail in a following paragraph.

The Nose and Postnasal Space.—The vestibule of the nose and the anterior tips of the lower turbinates may occasionally be photographed with simple equipment if the patient has large nares. Endoscopic equipment becomes necessary, however, for photography of the structures along the lateral nasal wall. This work has been done with the motion picture equipment developed for bronchoscopic photography (described on p. 762). The use of the camera developed by Clerf⁵ for mirror laryngoscopy as well as the one just mentioned has made photography of the postnasal space a possibility simply by reversing the laryngeal mirror and retracting the soft palate. The endoscopic camera may also be used to photograph the field seen through a Yankauer speculum. However, landmarks which are significant enough to make the picture taken through this speculum of value are not easy to include in still pictures of this area. Motion pictures with frequent reference to landmarks are thus more satisfactory than the stills.

The Larynx.—Excellent motion pictures of the larynx are now being obtained with many fundamentally different types of camera equipment. Simplified apparatus for routine case-recording of both the mirror and the direct laryngoscopic views of the larynx has been the object of most of the research in this field. Color film, either 16 mm. or 8 mm., at 16 frames per

second, is the accepted standard for this work. The simplest methods of mirror photography of the larynx employ a Ciné Kodak Special camera with a 2-inch or 3-inch lens. Illumination is obtained by directing the beam of light of a motion picture projector along the camera lens and onto the laryngeal mirror.^{6,7,8} The camera, mounted on a tripod, is placed directly in front of the patient and by means of the reflex finder in the camera the image in the laryngeal mirror is brought into focus.

For routine photography during direct laryngoscopic procedures using the Jackson-type laryngoscope, the additional illumination necessary for photography may be obtained by adding small 4 to 8-volt lamps to the periphery of the laryngoscope and overilluminating them during filming. The essential features of the photographic equipment consist of a lightweight camera, preferably with a reflex finder unless the reflex finder is placed in the optical system, an extension tube between the camera and the lens to secure proper focus and magnification, a 2-inch, 2½-inch, or 3 inch lens, and finally a rigid connecting tube by which the camera is secured to the laryngoscope to make the entire assembly a single unit (Fig. 556). Details regarding cameras, lenses, extension tubes, reflex finders, connecting tubes, and the modifications of the laryngoscopes themselves are so numerous that direct references to the original descriptions are necessary.^{9,10,11} A similar assembly for an 8-mm. film camera has also been described.¹² (Fig. 557).

Refinements of apparatus and the construction of more efficient lighting systems have produced photographic results which the simpler camera assemblies just described cannot approach. The equipment developed by Clerf⁵ for mirror laryngoscopic photography is notable for the extreme depth of focus it affords, giving great clarity to the entire laryngeal image, with exceptionally good color and texture. The apparatus (Fig. 558) is light and easy to operate. Special problems in physiology, pathology, and surgery have been illustrated through the use of other unique types of equipment. LeJeune¹³ (Fig. 559) and, later, Lierle,¹⁴ using suspension laryngoscopy, have devised equipment with which it is possible to photograph certain direct endoscopic surgical procedures. Pressman,¹⁵ using distal illumination direct laryngoscopy, has been able to study and illustrate laryngeal physiology through a

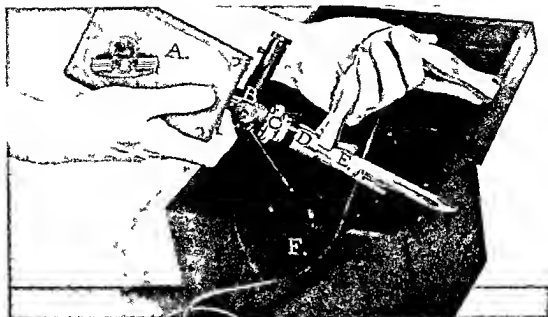


Fig 556—Camera, lens, and laryngoscope assembly for routine photography through the direct laryngoscope (A) 16 mm. magazine loading camera, index finger of right hand operates camera (B) reflex focuser, (C) f3.5 two-inch lens with special focusing mount (D) adapter sleeve fitting directly onto laryngoscope (E) photographic laryngoscope, (F) battery box (From Hohinger Paul H. and Mendeth H. W. *Ann Otol., Rhin. & Laryng.* 51)

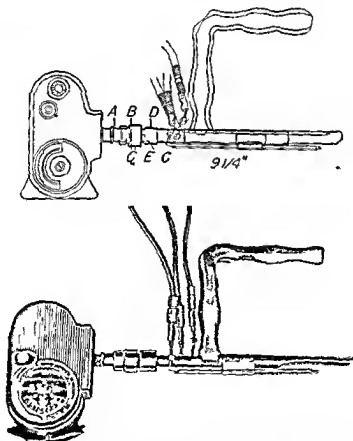


Fig 557—Camera, lens and laryngoscope assembly for laryngeal photography using 8 mm camera (A) f.25 universal focus lens, (B) No. 3 Leica f3.5 front lens, (C) extension tube, (D) collar for breathing holes, (E) breathing holes (From Solo, Adrian Fineberg, Nathan L., and Levene, George *Arch Otolaryng.* 30)

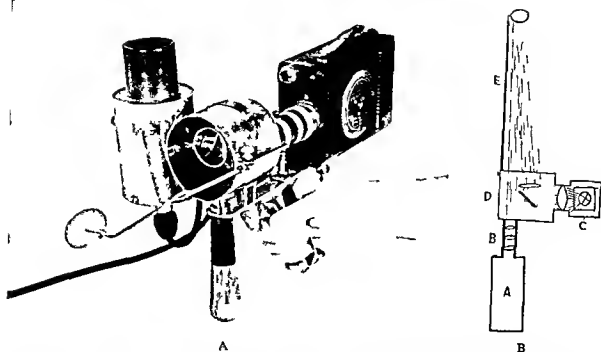


Fig 558 —A, Photographic unit designed by Clerf for auricular laryngoscopic photography B Diagram of unit (A) Magazine loading 16 mm camera (B) two-inch Taylor Hobson lens (C) 200-watt projection lamp (D) chamber supporting mirror and condensing lens (E) laryngeal mirror (From Clerf Louis H Arch Otolaryng 33)

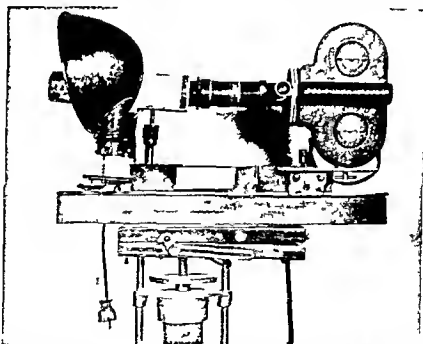


Fig 559 —Camera viewer extends on tubes, lens, and source of illumination mounted on tripod for laryngeal photography using suspension laryngoscopy (Courtesy of Dr Francis E Le Jeune)

motion picture record of the cords in various types of phonation and from these studies has introduced new concepts of phonation. Probably the most instructive films ever made from the standpoint of laryngeal physiology are those

of indirect laryngoscopy taken with a very high speed motion picture camera (4000 frames per second) developed by the Bell Telephone Laboratories¹⁶ (Figs 560-561). Analyses of the action of the entire larynx have been made with

this photographic equipment.¹⁷ The complete cycle of individual cord vibrations occurring with various vowel sounds at different pitches

effects of lesions of the larynx on the voice. Thus, the preoperative voice and corrections following various surgical procedures are

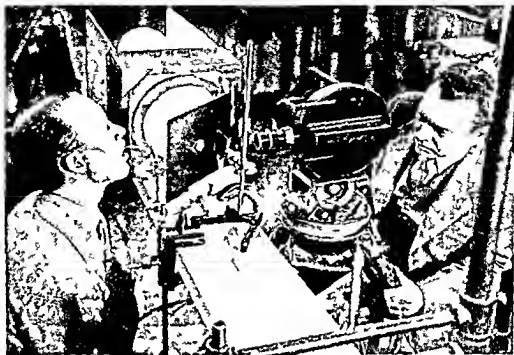


Fig. 560—Technic used by the Bell Telephone Laboratories for high speed motion pictures of the larynx by indirect laryngoscopy (4000 frames per second) (From Farnsworth D W Bell Laboratories Record 18)

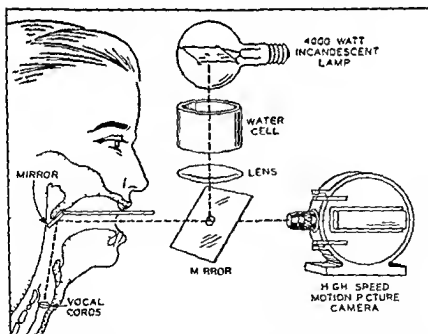


Fig. 561—Diagrammatic arrangement of equipment illustrated in Figure 560 (From Farnsworth D W Bell Laboratories Record 18)

has been recorded. Synchronization of the laryngeal films with the sound produced has been developed by Jackson¹⁸ to demonstrate the

recorded and an accurate correlation of the laryngeal picture with the sounds produced is obtained.

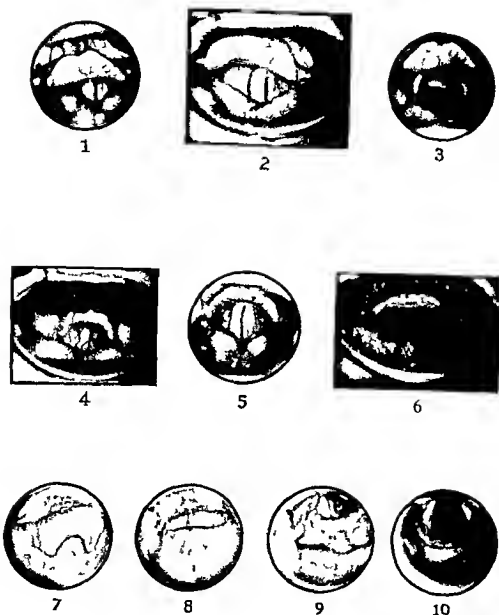


Fig 562 —Kodachrome photographs of the larynx taken with the camera illustrated in Figure 563

- 1 The larynx on phonation with an overhanging epiglottis. The vallecula and glosso-epiglottic folds are shown
- 2 Normal larynx on phonation with the entire mirror shown.
- 3 Normal larynx on inspiration.
- 4 Multiple papilloma of the larynx in a thirteen year old child
- 5 Papilloma along the left vocal cord of an eighteen year old girl
- 6 Extensive postcricoid carcinoma with invasion of the right cord and complete fixation of the right side of the larynx.
- 7 8 9, 10 Four steps in exposure of the larynx with the direct laryngoscope

The equipment designed for bronchoscopic and esophagosopic photography which is described in detail below has been adapted for both indirect and direct laryngeal photography. The field is proximally illuminated and the laryngeal image fills the height of the 16 mm frame. True axial illumination, proper masking, and the ability to view the field during finding and filming are the advantages of this equipment.

Interest in motion pictures of the larynx has been so keen that the progress of still

camera is used, the lens is a 4-inch focal length f 9.5 lens operated at f 11 to f 16 at 1/20 second for Kodachrome. It is mounted in a housing behind a hole in a 45 degree angle mirror, the mirror reflecting the light down the laryngoscope and onto the field from both the focusing lamp which shines through the wire filled flash bulb and from the flash lamp. A telescope permits continuous visualization of the field, magnified about seven times. Both the telescope and the camera are fixed in adjustment and are both focused upon the same plane in the

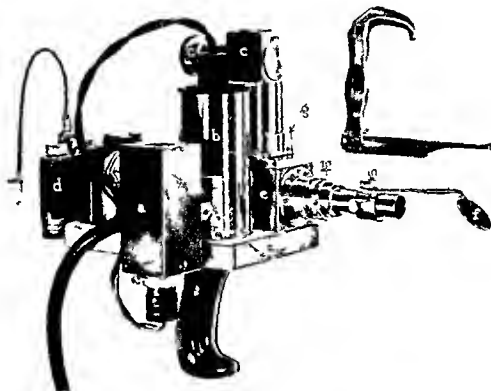


Fig. 563 —Flash camera for endoscopic photography (a) Focusing lamp housing (b) flash bulb housing (c) telescopic viewer (d) Leica camera (e) lens and 45° angle-mirror housing (f) laryngeal mirror (g) heated glass slide protecting lens and mirror (From Brubaker J. D. and Holinger Paul H. J. Biol. Photographic A. 10)

picture photography in this field has lagged behind the technical advances actually made in the still cameras. Lens systems, distal illumination, and proximal illumination equipment have been developed for them. Effenberger,¹⁹ using a lens system similar to a cystoscope, has presented fair results with the lens system technique. The distally lighted equipment employs the laryngoscopes developed for motion pictures with one of the 35 mm cameras. A proximally illuminated flash camera for either indirect or direct photography has been developed by Brubaker and Holinger⁴ (Fig. 563). A Leica

object space about 8 inches beyond the front of the instrument where either the direct laryngoscope or the laryngeal mirror may be attached. The whole equipment is moved by means of a handle to bring the object into focus. Since the point of focus is a fixed distance in front of the light mirror, the light intensity from the flash bulb will always be the same, an important factor when using Kodachrome (Fig. 562).

The Trachea, Bronchi, and Esophagus —The instrument developed for bronchoscopic and esophagosopic motion picture photography

uses the basic design of the proctoscopic camera described by Brubaker,²⁰ and by Garner, Nesselrod and Peerman.²¹ It is a unit consisting of a

may be attached.^{4, 22} (Figs 564, 565) Adequate illumination is obtained at the correct color temperature for Kodachrome Type A film

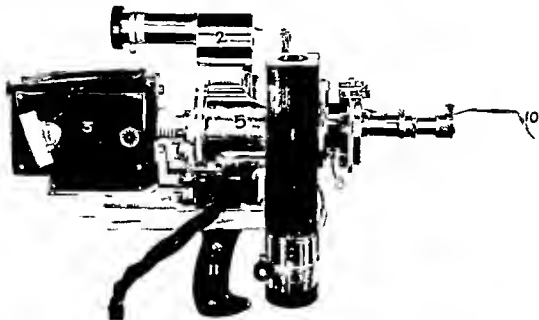


Fig 564 —Endoscopic motion picture camera arranged for taking mirror pictures of the larynx (From Brubaker, J D, and Holinger, Paul H. *J Biol Photographs* A 10)

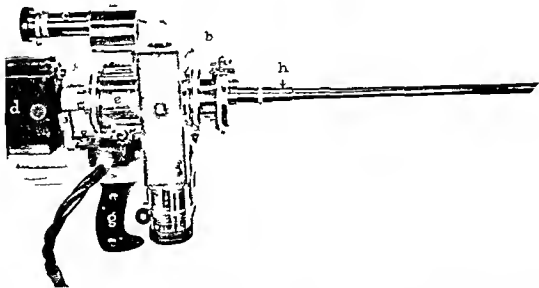


Fig 565 —Endoscopic motion picture camera with bronchoscope in place (a) Lamp housing (b) heated glass slide protecting lens (c) telescopic viewer (d) 16 mm magazine loading camera (e) lens housing (f) clamp for instantaneous detachment of camera (g) handle with trigger starting and stopping camera (h) sleeve for lengthening and shortening bronchoscope (From Brubaker, J D and Holinger, Paul H. *J Biol Photographs* A 10)

lamp, lens housing, movie camera, and telescopic eyepiece to which specially constructed endoscopic instruments, or the laryngeal mirror,

through the use of a 6 volt 18 ampere (108 watt) ribbon filament projection lamp which is mounted in a lamphouse at the side of the

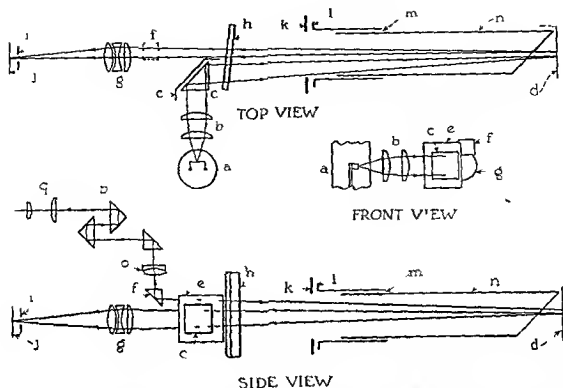


Fig 566 —Optical system of endoscopic movie-camera (diagrammatic) (a) Ribbon filament lamp, (b) condenser lenses (c) light prism (d) plane of sharp focus at object (e) septum and light shield (f) telescope prism (g) 3 inch focal length camera lens f 2.7, operated at f 4.0 (h) optically flat glass slide to protect optical parts (i) mask to shield film from flare from inside of endoscope (j) 16 mm motion picture film in camera (k) mask to absorb light flaring back from flange, (l) attaching flange of endoscopes (m) sleeve joint to allow adjustment of endoscope length (can be clamped), (n) laryngoscope bronchoscope or esophagoscope (o) telescope objective lens (p) erecting prisms (q) eyepiece of telescope

instrument The image of the center portion of the ribbon filament is directed along the axis of the scope by means of a condenser and a prism, the latter projecting into the lumen of the scope. The camera is a Bell and Howell 141-A, and is run at 12 or 16 frames per second. The lens, a Zeiss Tessar f 2.7, 8 cm (3½ inch) focus, operated at f 4, is mounted in a housing behind a removable heated glass slide. The slide prevents the condensation of moisture on the lens. A telescope which permits constant vision of the field for finding and focusing, as well as during the actual filming, is built into the optical system. A prism system makes the image erect and correct from side to side. Since the endoscopic field may be visualized at all times through the telescope, the endoscopes may be introduced attached to the camera. A trigger in the handle starts and stops the camera and at the same time increases the lamp voltage automatically from 6 volts to 9 volts during the actual filming.

The plane of critical focus is fixed in this camera, and the object is kept in correct focus

by movement of the camera backward and forward. Two fixed settings are provided, one for laryngeal and one for bronchoscopic or esophagosopic distances. Substitution of a laryngeal mirror for the direct laryngoscope makes mirror pictures of the larynx possible. By reversing the mirror the postnasal space may be photographed.

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AVIATION OTOLARYNGOLOGY

Introduction—Certain of the unavoidable conditions associated with flight in present-day aircraft may affect the ears, hearing, the nasal accessory sinuses, and other of the organs and functions generally considered to lie within the province of the otorhinolaryngologist. In general, these unavoidable conditions are barometric pressure variation coincident with ascent and descent, noise, which unfortunately seems

an inescapable concomitant of power and speed, and anoxia, which results from oxygen insufficiency. Anoxia is a problem only in the higher reaches of the earth's atmosphere, as it can be compensated for at least to an altitude of 33,000 to 35,000 feet by a specific remedy—supplementary oxygen.

Movement per se influences a portion of the body's physiological mechanism which has always been of utmost interest to the otorhinolaryngologist—namely, the vestibular apparatus. Motion at constant velocity is probably not perceived by the vestibular apparatus, but changes in rate or direction of motion, that is acceleration or deceleration, tangential or linear, are perceived by this structure and transmitted to the brain through its associated pathways.

To understand better the nature of the environment in which flight takes place, one must consider the physical characteristics of the atmosphere which surrounds the earth upon which we dwell. Reference to Figure 567 should furnish this necessary basic conception.

Variation in Barometric Pressure.—All of the closed gas or air-containing cavities of the body are affected by barometric-pressure change. During ascent, the density of the atmosphere restraining the structures of the body progressively decreases, consequently, any material capable of expansion will expand. Conversely, on descent, there is progressive compression of the same material. The degree of atmospheric-pressure change per unit of altitude above the earth's surface is not linear, but rather curvilinear (altitude pressure curve Fig 567). Thus, the effects produced by barometric pressure per linear unit of altitude are greater the nearer one approaches the surface of the earth.

Among the cavities of the body which can be affected by changes in barometric pressure are the middle ear and the nasal accessory sinuses.

Aero-Otitis Media.—Aero otitis media is an acute or chronic inflammation of the structures of the middle ear, produced by a differential between the pressure of the air in the middle ear and that of the surrounding atmosphere.

Etiology.—The dynamics of production of aero otitis media are as follows. During ascent, the barometric pressure outside the middle ear undergoes progressive reduction. Consequently, the pressure of the air trapped in the middle ear and contiguous structures becomes relatively positive, pushing the tympanic membrane (the most expandable element of the

cavity) outward, until the pressure differential reaches a sufficient degree to force open the eustachian tube, or is equalized by means of the sudden opening of the eustachian tube during the act of swallowing or yawning. The valvular action of the eustachian tube, except in abnormal conditions, offers little resistance

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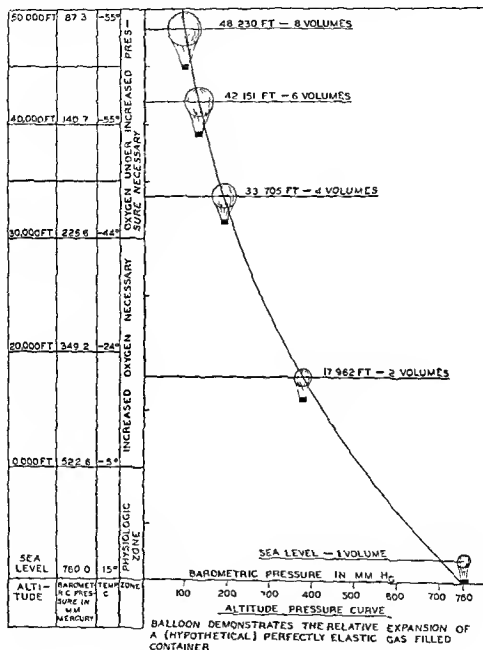


Fig 567—Characteristics of the atmosphere

to the outward flow. Consequently, difficulty during ascent is rare. During descent, however, the eustachian tube acts as a flutter valve, and as the outside atmosphere becomes denser, and the air in the middle ear relatively less dense (vacuum), a tight closure of the eustachian tube

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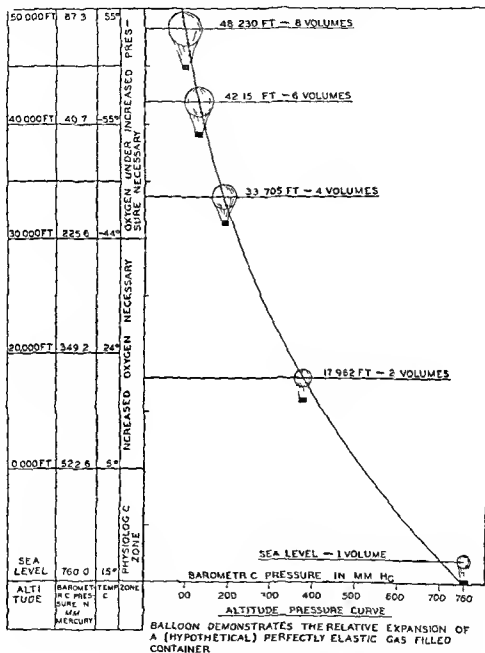


Fig 567—Characteristics of the atmosphere

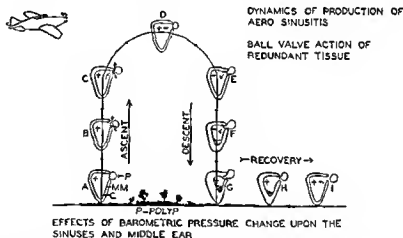
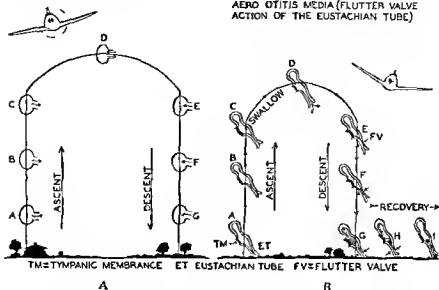
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FLIGHTLIKE EXCURSION OF A RIGID CONTAINER (SUCH AS A SINUS) WITH COMMUNICATION TO OUTSIDE ATMOSPHERE

DYNAMICS OF PRODUCTION OF AERO OTITIS MEDIA (FLUTTER VALVE ACTION OF THE EUSTACHIAN TUBE)



EFFECTS OF BAROMETRIC PRESSURE CHANGE UPON THE SINUSES AND MIDDLE EAR

C

Fig 568 —A, Demonstration of direction of flow during ascent and descent in the case of a rigid container with a communication to the outside atmosphere

B, Diagrammatic sketch of a flightlike excursion of the middle ear and contiguous structure. During ascent (A, B and C) the middle-ear pressure becomes relatively positive, pushing the tympanic membrane (TM) outward. After swallowing, pressure is equalized (D) and the tympanic membrane returns to normal position. During descent (E, F, and G) a flutter valve (FV) is formed. A relative vacuum causes the production of a serosanguineous exudate in the middle ear. Recovery (H, I) takes place after sufficient fluid is formed in the middle-ear cavity to equalize the pressure, thereby allowing the eustachian tube to open.

C, Flightlike excursion of a sinus demonstrating the ball valve effect of redundant tissue, such as a polyp (P) lying adjacent to the ostium. During ascent (A, B and C) air is forced out. Redundant tissue, such as a polyp, swells slightly due to decreased pressure and closes the ostium. Descent (E, F, and G). The redundant tissue acts as a ball valve. Relative negative pressure causes mucosa to swell and fluid is produced inside the cavity. Recovery begins when pressure is equalized (H).

nation discloses a minor degree of retraction of the drum head, especially the pars flaccida. There may be a slight inflammation of the drum head, usually localized along the handle of the malleus. Hearing threshold may be depressed

to a minor degree through the tone range, in the usual pattern of a conduction hearing loss. In the cases which have followed prolonged descent without ventilation, the symptoms and objective findings may be quite spectacular.

Excruciating pain may be experienced and in turn may be associated with extensive pathologic alteration of the drum and lining membranes. In severe degree, there may be punctate hemorrhages in the substance of the tympanic membrane, most often in the pars flaccida and along the posterior aspect of the handle of the malleus. During the first few hours sharp retraction is usually present and hearing is markedly depressed, according to the conduction pattern. As the relative vacuum must be compensated for, serosanguineous fluid is drawn from the mucous membrane lining the middle ear forming bubbles which, by their elliptical outline, may be identified shining through the tympanic membrane. Fluid blood may gravitate to the bottom of the cavity of the middle ear and form a definite line.

Diagnosis—Diagnosis is based upon a history of aural pain during exposure to barometric pressure change, oftentimes during an episode or just before an episode of pharyngitis or other upper-respiratory manifestation. In the light of proper history, retraction of the tympanic membrane, punctate hemorrhages in the membrane, and serosanguineous exudate seen through the membrane are diagnostic.

Differential Diagnosis—Aero otitis media must be differentiated from purulent and catarrhal otitis media. Differential diagnosis can be made on the basis of history and appearance of the drum head. Fever and leukocytosis are usually not present, or if present in aero otitis media, only to a very minor degree.

Course—In mildest degree the symptoms and findings are only transitory. In severe degree, the symptoms and findings may continue for a period of from seven to twenty-one days.

Treatment—From the standpoint of prophylaxis, those who have an upper-respiratory infection should not fly. If they must fly, they should not be subjected to great or rapid change in altitude. The structures about the eustachian orifice should be shrunk before flight, and means to keep them shrunken provided. The flyers should be warned to swallow or yawn voluntarily, and to move their lower jaws from side to side during descent. Children, and at times adults, are able to prevent ear blockage by sipping drinks or chewing gum. The Valsalva maneuver is a most efficient and popular method of ventilating the middle ear, but probably should not be used if infected materials can be forced into the ear.

To treat developed cases of aero-otitis media, immediate constant-pressure politization, employing a maximum of 2 pounds of pressure through a spray bottle containing a mild skriming medication, seems to be the method of choice. If possible, this procedure should be repeated every two hours or so during the initial stages. Then once or twice per day until ventilation is established. Heat applied to the ear supported by mild analgesics taken internally will frequently aid in the relief of pain.

Sequelae—Mild barotraumatic changes of the ear are usually reversible, and subside completely within a few days. The severe type associated with extensive hemorrhage into the tissue and the middle-ear cavity may be followed by organization of the blood, causing more or less hearing loss, and occasionally diplacusis. All except extremely rare cases, however, will resolve after a time.

Aerosinusitis—Aerosinusitis is an acute or chronic inflammation of one or more of the nasal accessory sinuses, produced by a barometric-pressure differential between the air inside the sinus and that of the surrounding atmosphere. It is commonly characterized by congestion or inflammation of the lining structures. Pain over the area of the sinuses is usually present. Mucosal or submucosal hemorrhage may occur. The condition results in temporary or at times permanent change in the mucous membrane lining the sinuses, depending upon the amount of barotrauma to which the individual has been subjected.

Etiology—The dynamics of production are somewhat similar to those of aero-otitis media. However, the check valve which prevents the free flow of air through the normally patent ostia during ascent and descent in the production of aerosinusitis is usually produced by the action of swollen and redundant tissue or polyps adjacent to or in the ostia. A ball valve produced by a globular mass of pus may be a causative agent. In the instance of a sinus with unobstructed ostia, no effect is produced by barometric-pressure alteration, as the air simply flows outward during ascent and inward during descent, thus constant equilibrium is maintained. During descent, however, pus or debris from an uncomplicated rhinitis can be pressed into a sinus, and would seem to offer an ideal opportunity for infection. Practically, however, this does not occur as often as might be ex-

pected, owing, it is believed, to the natural resistance of normal sinus epithelium

If the ostium is partially obstructed by means of redundant tissue, polyps, or debris the pressure gradient produced by altitude change can cause complete obstruction. Production of symptoms during ascent is relatively rare. On descent, however, the valve produced by a tissue flap may make pressure equalization between the sinus cavity and the ambient air impossible, and if descent is continued, the relative negative pressure (vacuum) inside the cavity will increase. If the relative negative pressure becomes great enough, the symptom picture of aerosinusitis will be presented (Fig 568 C).

Symptoms—The most common complaint following barotraumatic change in a sinus is excruciating pain occurring suddenly during descent, or shortly after completion of descent. It has at times been compared to the sting of a bee, or the sticking of a sharp point over the involved sinus. The frontal sinuses are most often affected, owing possibly to the length and anatomical relationship of the nasal frontal duct, as well as the relative volume of the sinus. The maxillary sinus is next in incidence. The other sinuses are undoubtedly affected under certain circumstances, but complaints referable to these sources are rare.

Diagnosis—Aerosinusitis must be differentiated from purulent and catarrhal sinusitis. This differentiation is aided by the elicitation of a history of pain over one or more of the sinuses following exposure to barometric pressure change. Epistaxis during or subsequent to such change is suggestive. Roentgenologic examination disclosing opacity or a degree of thickening of the lining membranes in absence of previous sinusitis is strongly indicative. Submucosal hematomas may be demonstrated in some instances in which the barometric pressure change has been great (20,000 to 40,000 feet). It must be borne in mind that aerosinusitis can occur in a sinus which is already infected, thereby altering the clinical picture of each of the entities.

Course—The milder cases are self limited. The more severe may run a course of a few days to a few weeks.

Complications—If the traumatized tissues do not become infected, recovery is usually rapid and unremarkable. If, as rarely occurs, the traumatized tissues do become infected, a severe

purulent sinusitis may result. Hemorrhage into the cavity of a sinus is not rare, if great pressure changes are involved. Submucosal hematomas or stripping of the mucous membrane away from the wall of a sinus may be seen roentgenographically in some of the more severe cases.

Treatment—Prophylaxis is similar to that of aero otitis media. If one must fly while experiencing an upper-respiratory infection, he should be advised to empty his nose of secretion before descent is begun. Ideally, in developed cases the patient should be reascended to the altitude at which obstruction occurred, his nose then emptied of secretion, his nasal mucous membranes shrunk, followed by a slow return to earth. During descent the Valsalva maneuver should be performed at times. Except in those cases developing in an altitude chamber, reascent usually is not feasible. Active treatment should be directed toward aiding equalization of pressure inside and outside the sinuses, and toward the restoration of normal ventilation. This involves the application of heat and mechanically moving the offending obstruction. Mild analgesics and sedatives may be used when necessary.

Aircraft Noise—The noise of present-day aircraft is intimately associated with power and speed. The intensity of such noise may reach a level of 120 decibels or more in certain of the frequencies. It is produced by a combination of engine explosions, propeller bum, slip stream effect upon structures, and sounds emanating from high-speed moving parts. Sound proofing, to a great extent at least, seems to be a function of the weight and bulk of the sound-proofing materials employed. Consequently, the speed, rate of climb, maneuverability, and carrying load of aircraft are directly affected by measures taken to decrease noise. In civilian aircraft a compromise can be reached, but in military aircraft protection must be obtained through the use of ear defenders (cotton plugs seem most popular), helmet protectors, and the screening afforded by carefully designed radio and interphone communication headsets.

Noise Deafness—The effect of noise is manifest in a temporary elevation of the hearing threshold, often in a notchlike pattern in the high tone range between the frequencies of 2000 to 6000 cycles. The rise in threshold is usually in a frequency area much higher than the frequencies of greatest intensity produced by aircraft. The exact cause of this phenomenon

is not as yet completely explained. Hearing loss produced by aircraft noise is, except in rare instances, reversible, and, as the amount of loss is related to the intensity of the noise and the length of the exposure time, seems to fit into the usual conception of fatigue. Recovery time, as in other types of fatigue, bears a definite relationship to the intensity and exposure time.

If the noise intensity is great, the exposure time prolonged, and the acoustic mechanism of the subject vulnerable, notchlike elevation of the threshold may become permanent. Permanent notching has been reported to have occurred in some individuals after a period of from 200 to 2000 hours of flight in high powered aircraft. Likewise, it is known not to have occurred in individuals with as much as 20,000 hours in the air. Thus, the vulnerability of the hearing mechanism of the individual becomes an important consideration. The reason for this predisposition is as yet undetermined. In evaluating the hearing loss of any aviator, the examiner must always realize that age, previous ear diseases, and noise trauma, other than that produced by flight, must be considered.

Airsickness—Airsickness is a form of motion sickness, resulting from the random motion of aircraft in flight or aerobatic maneuvers. Psychogenic elements in certain individuals may condition an individual's threshold of tolerance to motion. It is characterized by indisposition, pallor, sweating, change in respiratory pattern, gastric awareness, nausea, and vomiting.

Etiology—Airsickness may be produced by motion of the aircraft or emotion of the subject during flight. In most instances, it is a mixture of two elements. The motion element is provided by random movements, consisting of bumping, lateral rotation, pitching, and yawing. The movements in most instances take place about a definite center of rotation, which varies in position with each type of aircraft, but is usually situated near the center of the main wing. The movement other than that produced by aerobatics is due to rough air resulting from thermal currents. Faulty pilot technique and poor trimming or loading of aircraft may be a cause in relatively smooth air.

The psychogenic element in the production of airsickness is usually considered to be a manifestation of fear of flight. At times reaction to peculiar odors or lack of adequate ventilator may act as a provocative.

Pathology—Hyperactivity or imbalance of the vestibular apparatus may be a cause. However, in many instances, vestibular reactions of the individual are within normal range. Airsickness occurs often in those with a psychoneurotic background.

Diagnosis—History of gastric awareness, nausea, and vomiting during flight is usually sufficient. In most cases, history of other forms of motion sickness can be elicited.

Treatment—Flight in rough air, or after food or alcoholic excesses, and fatigue should be avoided. Individuals who are susceptible should attempt to seat themselves as near as possible to the center of rotation of the aircraft. Hyoscine or hyoscine-like drugs in small doses taken one to two hours before flight seem to offer more protection than other remedies.

Prognosis—Those individuals in whom the motion element is the predominating factor tend to adapt well and after a few flights are no longer affected. On the other hand, those in whom the emotional element is strongest do not adapt well until the psychogenic cause is removed.

Sequelae—Persons in whom the motion factor predominates as the causative agency are usually free of symptoms shortly after return to earth. Those in whom the psychogenic element is strongest may complain of nausea and headache as long as twenty-four hours or longer after cessation of their aerial journey.

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FOCAL INFECTIONS IN RELATION TO THE NOSE, THROAT, AND EAR

Although the problem of causative foci of infection had been investigated for many years by those workers engaged in the study of nephritis, it was not until 1912, when Billings published his paper on chronic foci of infection that general interest was aroused. The term "focal infection" is usually applied to a localized infection which gives rise to symptoms in other parts of the body. There are acute and chronic foci. The term "chronic focus" is usually applied to silent localized infection.

The nose, throat, and ear are common sites of foci infection, both acute and chronic. Crypts of the tonsils and lymphoid tissue, in the pharynx, base of the tongue, or nasopharynx may be the site of foci, as may the mucous membrane of the sinuses, the middle ear, and the mastoid cells. In infections of the maxillary sinuses, the underlying cause may be an abscess of an upper molar tooth.

A clinical example of an acute focus of infection in the nose, throat, or ear is seen in cases of mastoiditis complicated by a thrombosis of the lateral sinus. Here it is known that infected thrombi are scattered over the body and bacteria can be cultured from the peripheral blood. In other cases of focal infection, the changes in the body may be caused not by the actual bacteria, but by absorbed toxins from the bac-

terial growth in the focus, or an allergic reaction by the body to those toxins. As an example, the rash of scarlet fever and the early intoxication are presumably caused by circulating toxins while the complications such as adenitis, meningitis, or arthritis are due to an invasion of the tissues by the bacteria themselves.

Crowe has shown that there is a thrombosis of the adjacent small veins in chronic infection of the crypts of the tonsils with minute infected thrombi being fed into the blood stream, and others have shown that multiple abscesses are located in the thickened chronically-infected mucous membrane of the sinuses.

The bacteria from these foci or their toxins may cause destructive processes in other parts of the body. The removal of the focus may stop the destructive process, though the normal function of these parts may not return if the destructive process has been irreparable.

Conditions which may be associated with foci of infection are chronic arthritis (infectious type), rheumatoid arthritis, myositis, neuritis, fibrositis, acute glomerular nephritis, erythema nodosum, pyelitis, phlebitis, and inflammatory lesions of the eye (iritis, uveitis, keratitis, and choroiditis). Patients suffering from the effects of chronic foci of infection may show general debility, loss of weight, secondary anemia, fatigue, and muscular weakness.

As the patient is frequently unaware of any contributory infection, it behooves every otorhinolaryngologist, when studying persons suspected of having foci of infection, to take a careful history and to make a painstaking examination of the ears, the nose, the sinuses (by transillumination and roentgen ray), the posterior nares and nasopharynx (with a nasopharyngoscope) (in small children a general anesthetic may be required), and the tonsils and lymphoid tissue of the pharynx and the base of the tongue.

Infected mucous membranes of the sinuses and infected tonsils should be removed surgically as should an infected mastoid. The infected lymphoid tissue of the nasopharynx should be removed surgically and by the use of radium or roentgen rays.

In general, it is considered a favorable sign if, after the removal of foci of infection, the underlying condition flares up, such as in arthritis the joints become more painful or changes in the urine of nephritis become more

pronounced. This flare up is followed by a gradual secession and clearing-up of the disease.

Chronic systemic diseases, such as tuberculosis, when associated with chronic infectious foci, may be benefited by their removal.

Acute foci should be removed only when the acute condition is subsiding, usually from two to four weeks after the onset, as in acute tonsillitis followed by acute glomerular nephritis.

The use of the sulfa drugs and recently tyrothricin and penicillin have aided greatly in the destruction of pathogenic bacteria in both generalized and localized infections and have promoted the rapid healing of infected fields requiring surgery.

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CHEMOTHERAPY IN OTOLARYNGOLOGY

THE SULFONAMIDES

During the past ten years great strides have been made in the treatment of suppurative diseases of the ear, nose, and throat by means of chemotherapy. The first chemotherapeutic agent given to the medical profession dates

back to the fourteenth century when mercury was brought out for the treatment of syphilis. In the following, or fifteenth, century, quinine was given to us for the treatment of malaria. There was nothing further done along this line of therapy for almost five hundred years until Ehrlich in 1909 introduced salvarsan for the treatment of syphilis. In this same year, Gelmo discovered *sulfanilamide* but did not apply it clinically and it remained for Domagk to notice its potency in combating hemolytic streptococcal infections in mice. It was first put on the market and used by the profession in 1935, and within a year or two was the standard and accepted treatment for practically all diseases of infectious origin. It works especially well in streptococcal infections and to a lesser degree against other organisms. In 1937 another great drug came into use, *sulfapyridine*. It was found that this drug worked exceptionally well against pneumococcus upon which *sulfanilamide* had very little effect. One bad feature of this drug is that it is much harsher on the kidneys than is *sulfanilamide* and it has produced many serious kidney complications. In 1938 *sulfathiazole* was produced, which works very well against staphylococcus and is not nearly as toxic as *sulfapyridine*. In 1939 another great drug was given to the profession—namely, *sulfadiazine*. This drug works well against streptococcus, pneumococcus, and staphylococcus, it is just as effective, if not more so, as the other drugs and is much less toxic. Within the past year another sulfa drug, *sulfamerazine*, has come on the market. It acts very much the same as *sulfadiazine* and works well against streptococci, staphylococci, pneumococci, and gonococci. It is supposed to be more slowly excreted by the kidneys, and thus smaller or less frequent doses are necessary to produce and maintain therapeutic concentrations in the blood. More time will be required to study this new drug before it can be used in place of *sulfadiazine*.

Sulfadiazine is the drug of choice at the present time for the following reasons:

1. It works well against all organisms.
2. It is readily and slowly absorbed from the gastrointestinal tract.
3. It has fewer toxic manifestations than any of the other sulfonamides.
4. It can be carried in the blood in much higher concentration than can any other of the sulfa group.
5. It diffuses readily into all fluids of the body, attaining a concentration in the cerebrospinal fluid equal to 50 per cent of the blood concentration.

cavities of radical mastoidectomies in which skin has not been grafted, it seems to work very well. Aqueous solutions of a sulfonamide compound may be instilled into the mastoid antrum after irrigation.

In bronchopulmonary suppurations, treatment by suction irrigation of 5 per cent sodium sulfathiazole solution into the bronchi has proved successful in many cases. This procedure may be repeated every two or three days.

In the active treatment with sulfonamides, large doses of one of the compounds are administered to patients with severe nose, throat, or ear infections. In milder infections it is questionable as to whether or not chemotherapy should be used. To employ the sulfa drugs intelligently one must be familiar with

their toxic reactions and must weigh in the balance the dangers of these toxic reactions and the dangers of the infection. When the infection is severe, such as in sinus thrombosis, erysipelas, meningitis, cellulitis, deep infections of the neck, Ludwig's angina, periesophageal abscess, and larynotracheal bronchitis, the patient should be given large doses of the drug at once without consideration of the toxic reactions since conditions of high mortality rating are being dealt with.

Dosage of a sulfonamide depends on many things, such as whether the drug is given prophylactically or for an active infection, whether the disease is serious or mild, whether the patient is an adult or child, large or small. The prophylactic dose for adults as a rule is 15

SULFONAMIDE THERAPY

Dosage for Adults

Drug	Disease	Route	Initial Dose	Subsequent Doses	Duration of Treatment
Sulfanilamide	Severe beta hemolytic streptococcal infections	Oral	90 grains (6 gm)	$\frac{1}{2}$ initial dose every 4 hrs day and night	Continue until temperature is normal for 72 hours then taper off the dose
Sulfapyridine	Pneumococcal pneumonia	Oral	60 grains (4 gm)	$\frac{1}{2}$ initial dose every 4 hours day and night. Give plenty of water.	Continue until temperature is normal for 72 hours then taper off the dose
Sulfathiazole	Severe staphylococcal infections such as cellulitis lymphangitis acute osteomyelitis	Oral	60 grains (4 gm)	$\frac{1}{2}$ initial dose day and night. Adequate amount of water. Alkalize urine.	Continue until temperature is normal for 72 hours
Sulfadiazine	Severe pneumococcal, hemolytic streptococcal staphylococcal and meningococcal infections	Oral	90 grains (6 gm)	$\frac{1}{2}$ initial dose day and night. Adequate amount of water.	Continue until temperature is normal for 72 hours then discontinue drug
Sodium sulfadiazine	Any of the above severe infections when it is necessary to saturate the bloodstream in a short time	Intravenous	50 cc of 5% solution	Repeat initial dose if necessary. Then 1 gm sulfadiazine by mouth every 4 hrs day and night.	If drug cannot be taken by mouth may be given intravenously every day to keep the concentration up to desired level.

Dosage for Infants and Children

Drug	Disease	Route	Initial Dose	Subsequent Doses	Duration of Treatment
Sulfanilamide	Beta hemolytic streptococcal infections	Oral	7/10 grains per lb (0.1 gm per kg)	$\frac{1}{2}$ initial dose every 4 hrs day and night	Continue until temperature is normal for 72 hours
Sulfapyridine	Pneumococcal pneumonia	Oral	1 grain per lb (0.15 gm per kg)	$\frac{1}{2}$ initial dose every 4 hrs day and night	Continue until temperature is normal for 36 hours
Sulfathiazole	Severe staphylococcal infections	Oral	15 grains (1 gm) for each 11 lbs (0.2 gm per kg)	$\frac{1}{2}$ initial dose every 4 hrs day and night	Continue until temperature is normal for 72 hours, then taper off
Sulfadiazine	All above infections	Oral	15 grains (1 gm) for each 15 lbs. (0.15 gm per kg)	$\frac{1}{2}$ initial dose every 4 hrs day and night	Continue until temperature is normal 48 hours
Sodium sulfadiazine	All above infections when it is necessary to saturate the blood stream in a short time	Intravenous	15 cc of 5% sodium sulfadiazine	Repeat daily if drug cannot be taken by mouth	Discontinue as soon as drug can be taken by mouth

grains (1 gm) every four hours. In active and serious infections the dosage is much larger, to get the proper amount in the blood stream as soon as possible, 60 to 80 grains (4 to 5 gm) are given for the first dose, then 30 grains (2 gm) every four hours for two or three doses, cutting down to 15 grains (1 gm) every four hours day and night. Administration of the medicament throughout the night is important. Once the blood level stays at 10 to 20 mg per 100 cc of blood it can easily be maintained at this level with small doses. The doses of sulfamerazine are about the same as those for sulfadiazine. However, since sulfamerazine is supposed to be more slowly excreted by the kidneys, it is claimed that doses given every eight hours are sufficient to keep the blood concentration at the desired level. In very sick patients in whom it is desirable that the sulfa drug reach the blood stream as soon as possible, 50 cc of 5 per cent sodium sulfadiazine solution is given intravenously, in fifteen to twenty minutes the drug will be present in the blood. If the desired concentration is not obtained following the first dose, the intravenous injection may be repeated in four hours. Thereafter

the patient is given 15 grains (1 gm) every four hours by mouth. It is important that the patient be given small doses of the drug for a week or ten days after the symptoms have disappeared.

The dosage for children is reduced according to the weight of the child. The average initial dose in severe infections is about 0.1 gm per kilogram of body weight or 7/10 grains per pound, and the subsequent dosage is $\frac{1}{2}$ or $\frac{1}{4}$ of the initial dose administered every four hours day and night.

The question has often been asked, "Is it necessary to obtain a certain blood level?" Some patients seem to do well with a low level but most do better when the concentration is high. I believe that the concentration should be over 5 mg per 100 cc of blood with most of these drugs but with sulfadiazine from 10 to 20 mg. In serious cases I always feel safer if it is much higher, in two cases of type III pneumococcal meningitis it was raised to 40 and 45 mg per 100 cc of blood and both patients recovered.

The tables of dosages shown above and on the preceding page may be found useful.

Whenever the sulfonamides are given the physician must be on the alert for toxic reactions. Urinalysis, blood counts, and estimation of the blood concentration should be done every two or three days and in meningitis cases the spinal fluid is also examined for concentration.

Summary—It should be borne in mind that the sulfonamides do not take the place of surgery. When surgery is indicated it should be performed first, then the drugs administered. When these drugs are indicated in serious infections they should be given early and in large doses to bring the blood concentration up to the desired level in as short a time as possible. When sulfanilamide, sulfapyridine or sulfathiazole are used the concentration should be from 8 to 12 mg per 100 cc of blood, but when sulfadiazine is used it should be between 15 and 20 mg. The blood concentration may be raised as high as 40 or 45 mg in severe cases. In extreme cases the sodium salt should be administered intravenously, it shows up in the blood in about fifteen minutes.

All physicians prescribing the sulfa drugs should be familiar with the toxic reactions that may follow their use. But in severe infections the drugs should be used boldly without too much fear of the complications as only about 10 per cent of the patients receiving them suffer any complicating reactions. Plenty of fluids should be given and the blood concentration should be maintained.

Conclusions.—(1) The sulfa drugs should not be used when the dangers of toxicity are greater than the dangers of the disease. (2) When indicated, large doses should be given boldly early in the disease. (3) If no improvement is noted in seventy-two hours the drug should be discontinued. (4) Estimation of the blood concentration, blood count, and urinalysis should be made every forty-eight hours. (5) The sulfonamides are a valuable adjunct to surgery but are not to take the place of surgery.

If the desired effect is not produced by the drug, one should ask himself the following questions:

1. Is the diagnosis correct?
2. Are there any complications?
3. Is the patient getting a sufficient amount of the drug?
4. Is there an overwhelming infection, when pus is present, in which surgery will help?

PENICILLIN

Penicillin, the newest of the chemotherapeutic drugs, is effective in cases in which the sulfa drugs fail to kill infection. It was discovered by Alexander Fleming at the University of London in 1929, and it was not used clinically until about twelve years later. It remained for the Oxford investigators in 1940 (Chain, Florey and others) to prepare a highly concentrated and active preparation of penicillin suitable for clinical use. Penicillin is obtained from molds and is produced in very small quantities, 1 cc of the fluid produces only 2 units of penicillin. It is relatively nontoxic and at the same time bactericidal under certain conditions. It is not related to nor does it behave like any of the chemotherapeutic substances now in use. It is not hemolytic. It is highly soluble. It does not behave as a detergent. Its action is not inhibited by the presence of para-aminobenzoic acid nor by the products of tissue destruction.

Penicillin is far superior to any of the sulfonamides in the treatment of *Staphylococcus aureus* infections with or without bacteremia, including acute and chronic osteomyelitis, cellulitis, carbuncles of the lip and face, pneumonia, empyema, and infected wounds and burns. It is also extremely effective in the treatment of hemolytic streptococci, pneumococci, and gonococci infections which are resistant to the sulfonamides. It has not been found effective in the treatment of subacute bacterial endocarditis.

Penicillin does not enter the meningeal spaces or spinal fluid following intravenous or intramuscular injections, therefore in treatment of meningitis it must be given directly into the spinal fluid as well as intravenously. In empyemic cavities the drug must be placed directly into the cavity. Intravenous or intramuscular administration alone is useless in these cases.

In congenital syphilis, treatment with penicillin shows great promise. In fifty per cent of the cases followed for twelve months after treatment, the patients appear physically normal and reactions to serologic tests are negative. In the treatment of early syphilis in adults, the immediate results have been very encouraging as far as the serologic reactions and the physical condition of the patients are concerned, but it will take time to determine the actual end results. In tertiary syphilis the response has not been as good. Recently some encouraging

work has been done with penicillin in combination with the hypertherm therapy in patients in the late stages of the disease

Contrary to the belief of two years ago that penicillin was ineffectual in the treatment of acute bacterial endocarditis it has proved most effectual if given in large doses

Mode of Administration—Penicillin may be given intravenously every four hours continuous intramuscular injection may be employed, it may be applied topically, or it may be given all ways at the same time. Some patients tolerate intramuscular injections better than intravenous and vice versa. Penicillin administered orally in combination with a buffer salt (usually sodium citrate) has recently been found to have the same efficacy as that given by the intramuscular or intravenous routes. Topical application includes local injections into the pleura, the pericardium, the joints, and the subarachnoid space. In the treatment of meningitis, penicillin has been injected directly into the subarachnoid space, in empyema into the pleural cavity and in surface burns of limited extent, it has been applied locally.

Dosage—Dosage is graded in Oxford units. The amount of the drug given varies tremendously from one case to another. There is also great variation in the total amount given with each injection, the interval of time between injections, and the total duration of the treatment. In the beginning it was common practice to give 5000 units intravenously every four hours day and night, i.e., 30 000 Oxford units every twenty-four hours. This amount was found to be adequate in some infections but was totally inadequate in others. The dosage often varies from 1000 to 25 000 units per hour depending on the kind and severity of the infection. The question of adequate or optimum dosage has not been clearly defined as yet. The objective in treatment should be the maintenance of a sufficient concentration of penicillin in the blood to inhibit completely the growth of the individual infecting organism. One method of assessing adequate dosage is by the signs of clinical response. These must be followed with great care and should include not only the response of the temperature and pulse rate but also the change in constitutional symptoms, the results of the blood culture, and the effect on the local infection.

To obtain the best results it is necessary to inject penicillin continuously or at frequent

intervals for at least seven to fourteen days or longer.

DOSE SCHEDULE

1 In serious infections due to hemolytic streptococcus, *Staphylococcus aureus* or pneumococcus with or without bacteremia, an initial dose of 15 000 to 20 000 Oxford units should be given with continuing dosage as follows: (a) 5000 units every hour injected into the tubing of an infusing intravenous set, or (b) constant intravenous injection of a solution at a rate designed to deliver 5000 to 10 000 units per hour. Occasionally larger doses are needed. After the temperature is normal the total dose in a twenty-four hour period may be reduced one-half but the treatment should be continued for at least seven days after temperature has become normal.

2 In chronic infections such as infected compound fractures or septic infections of soft parts the initial dose of 10 000 units is given followed by 15 000 units every three hours with local treatment as indicated.

3 In sulfonamide resistant gonorrhea 10 000 units are given every three hours for 12 doses, or the total dosage may be given in a single injection of 100 000 Oxford units employing beeswax or sesame oil as a vehicle.

4 In empyema penicillin in isotonic salt solution should be injected into the cavity after aspiration of pus or fluid. This should be done once or twice daily using 30 000 or 40 000 units.

5 In meningitis 10 000 units of penicillin in isotonic salt solution should be injected into the subarachnoid space or intracisternally once or twice daily. Penicillin should also be given intravenously or intramuscularly at same time.

6 In pneumonia 60 000 to 90 000 units a day are given for three to seven days.

7 In the treatment of infants with congenital syphilis penicillin is as yet in the experimental stage but it is safe to give 40 000 units per kilogram of body weight divided into 60 intramuscular injections, given every three hours for one week. In the treatment of early syphilis in adults approximately 3 000 000 units are given in divided doses over a period of one week.

8 In bacterial endocarditis 10 000 000 to 30 000 000 Oxford units are administered.

9 The oral dose is several times greater than the intramuscular—in general 50 000 to 100 000 units given every two or three hours until improvement occurs.

Absorption and Excretion—Penicillin is rapidly absorbed when given intravenously and slowly absorbed after subcutaneous injections. Following intravenous injection there is a high initial concentration of penicillin in the blood plasma; however, the drug fails to penetrate the red cells in significant amounts. The average concentration in red cells is only 10 per cent of that in the plasma.

Penicillin is rapidly excreted by the kidneys. This necessitates frequent administration when given intravenously or intramuscularly. There is good evidence that it is excreted by the liver also since it can be found in the bile in higher concentration than in the blood stream.

Toxicity and Reactions—The administration of penicillin is attended by very few toxic reactions. Occasional chills and fever were noted in twelve cases in a series of 500. Skin eruptions occurred in fourteen of the 500 cases, thrombophlebitis at site of injection in nineteen. Other toxic manifestations include headache, a throbbing tingling sensation in the testes, and pains in the muscles. These last only a few hours.

Summary and Conclusions—From the study of 500 cases of infection in which penicillin was used therapeutically the following conclusions are justified.

Penicillin is a remarkably potent antihacterial agent which can be given orally in combination with a buffer salt, intravenously, intramuscularly, or topically.

Following intravenous or intramuscular injection, penicillin is excreted rapidly in the urine, so that in order to obtain an adequate amount of potent material in the circulating blood and tissues it is necessary to inject the preparation continuously or at frequent intervals, that is, every three to four hours.

Penicillin has been found to be most effective in the treatment of staphylococcic, gonococcic, pneumococcic, and hemolytic streptococcic infections. Its effect is particularly striking in sulfonamide resistant gonococcic infections. It shows great promise in the treatment of syphilis.

While the dosage schedule calls for additional investigation, it seems clear that the average patient receiving intravenous or intramuscular injections for serious staphylococcic infections, requires a total of between 500,000 and 1,000,000 Oxford units and the best results have been observed when treatment is continued for at least ten days to two weeks. At least 10,000 units should be given every two to three hours at the beginning of treatment, either by continuous intravenous injection or by interrupted intravenous or intramuscular injections.

Patients with pneumococcic pneumonia frequently recover after having been given 100,000 units over a period of three days. Penicillin is especially important in sulfonamide resistant pneumococcic infections. It may be necessary to give between 60,000 and 90,000 Oxford units daily for four to seven days to obtain a maximum effect.

In the treatment of empyema or meningitis it is advisable to use penicillin topically by injecting it directly into the pleural cavity or the subarachnoid space.

Toxic effects are extremely rare. Occasional chills with fever, or headache and flushing of the face have been noted. Urticaria has been reported and thrombophlebitis at the site of injection has been described.

As in the use of the sulfonamides, it must be remembered that penicillin is not to take the place of surgery, but rather to act as an important adjunct to it. In the administration of penicillin it should be borne in mind that the drug is rapidly eliminated from the body completely in two hours. Its effects are accomplished only by the drug coming into direct contact with the organisms.

DANIEL S. CUNNING

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ANESTHESIA

General Considerations—With inhalation anesthesia, the fields of the otolaryngologist and of the anesthetist frequently overlap. For this reason the *insufflation*, *intravenous*, and *local* methods are more often applicable. Furthermore, the anesthetist depends largely on special positions of the head to keep the upper respiratory tract patent. But in otolaryngologic surgery the position of the head must usually be determined by the surgeon, with the result that dependence must be placed more frequently on the *endotracheal* tube to prevent respiratory obstruction. This is true whether anesthesia is produced by inhalation or by the intravenous route or by avertin in combination with local or other methods of administration. For operations on the nose and on the ear, anesthesia usually need not be deep, and the less potent and less toxic anesthetic agents may be used. For procedures involving the larynx and bronchial tree it is often desired to suppress reflexes which arise from stimulation of these regions, and to do this either local anesthesia or deep general anesthesia is necessary.

Preliminary Medication—Good preliminary medication is essential to the success of anesthesia. In patients who are to have a local anesthetic, this should produce sedation and some

degree of analgesia, and should afford protection against the convulsions that may be caused by an overdose of the local anesthetic. Preliminary medication may also be used to produce amnesia if desired. In those who are to have general anesthesia it is used to produce sedation, to reduce reflex activity, and to prevent undesirable by-effects of certain anesthetics, such as laryngeal spasm, vomiting, and excessive secretion of mucus.

Morphine and pantopon produce sedation and mild analgesia, and reduce reflex activity. In case of sensitivity to morphine, *demerol* may be used. *Dilaudid* produces less sedation and less reduction of reflex activity, and is more specifically analgesic. *Barbiturates*, such as *pentobarbital sodium* and *seconal*, produce sedation, lessen the vomiting after inhalation anesthesia, and also protect against the convulsions which may be caused by local anesthetics. For this reason one of the *barbiturates* should always be used before local anesthesia. *Scopolamine* produces amnesia, dries the mucous membranes, and counteracts the tendency of morphine to produce respiratory depression, nausea, and vomiting. For its effect on respiration the dose should be 1/25 that of morphine. Before local anesthesia the dose should be very small (0.2 mg or 1/300 grain), otherwise the release of inhibitions which it causes may be troublesome. If this occurs, the patient can usually be quieted by an additional dose of morphine given intravenously. *Atropine* also dries the mucous membranes, but probably less than *scopolamine*. It also counteracts the nausea and vomiting produced by morphine. It should always be given before pentothal to check the laryngeal spasm, hiccoughing, coughing, and sneezing (parasympathetic effects) which that drug may help to produce. But since atropine increases the metabolic rate and reflex activity, *scopolamine* is to be preferred when there is a choice between the two. *Avertin* in small doses produces amnesia and sedation, and in larger doses produces sleep, with varying degrees of analgesia. It is not safe to increase the dose with the intention of producing complete surgical anesthesia.

Timing—The opiates and the barbiturates should be allowed at least a half hour to take effect. It is well to give most patients a barbiturate by mouth about one and a half hours before operation, and morphine and *scopolamine* or morphine and *atropine* hypodermic-

ally one hour before operation. If the medication must occasionally be given within the last half hour, *nembutal C* may be used by mouth or *phenobarbital sodium* hypodermically, and morphine and *scopolamine* may be given intravenously. The doses of morphine and *scopolamine* may be the same as those which would be given hypodermically, and administration should take at least two minutes.

Doses—With all sedative drugs the doses should be ordered individually for each patient. Larger than average doses may be used for those between sixteen and thirty years of age, for patients with hyperthyroidism, fever, or chronic alcoholism, and for those who are of very muscular build or who are unusually apprehensive. Smaller doses, on the other hand, should be prescribed for the elderly, the weak, the debilitated, the markedly anemic, and for those with hypothyroidism. If a mild sedative effect from morphine is desired in children, they may be given 1/60 grain (1 mg) per year of age.



Fig. 569—Tube for oral insufflation of ether vapor (two thirds actual size) (Gwathmey)

To dry the mucous membranes before ether and to give some degree of amnesia, children up to six months of age may be given 1/1000 grain (0.06 mg) of *scopolamine*, 1/600 grain (0.1 mg) may be given to one-year-olds, and increasing doses may be administered with increasing age, up to 1/150 grain (0.4 mg) for sixteen-year olds.

Inhalation Anesthesia—Methods—The open mask, with ether or vinyl ether, is suitable mainly for brief operations that may be performed during emergence from anesthesia, such as myringotomy and adenoidectomy.

Anesthesia may be maintained indefinitely by the insufflation method. Ether may be insufflated through a curved metal tube (Fig. 569) placed in the corner of the mouth, or, better, it may be insufflated into the pharynx by a catheter passed through the nose or by a specially-designed tube (Fig. 570) placed in the mouth. Maintenance of prolonged anesthesia by insufflation of gases into the pharynx while the

mouth is kept open is much more difficult because of the rapid dilution of the gases with air. A special nasal mask should be used for this purpose (Fig 571)

either case, the tube should be of as large diameter as the respiratory tract will comfortably accommodate, and dead space should be reduced to the minimum. It may be connected

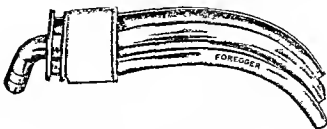


Fig 570—Tube for oropharyngeal insufflation of ether vapor (Buettner). The vapor is blown in through the tube. The patient breathes through the troughs.

The *closed* method with carbon dioxide absorption and with the ordinary surgical inhaler is not often advantageous for otolaryngologic work, since the mask covers both mouth and nose, and the mask strap usually covers the ears (Fig 572).

The various *endotracheal* techniques are the most useful methods of producing general anesthesia for this type of surgery. General anesthesia is first induced, or local anesthesia of the pharynx and larynx. There are then two commonly used methods available for intu-

into a semi open system (Fig 575), in which the endotracheal tube forms one arm of a T, a tube from a source of ether vapor forms a second arm, and the third arm is freely open to the air so that the patient may inhale and exhale through it. Or by means of a suitable adapter, the tube may be made a part of a closed system with carbon dioxide absorption (Fig 576). In this case it may be advisable to insure against any communication between the respiratory tract and the room air by placing packing around the tube in the pharynx, or by an in-

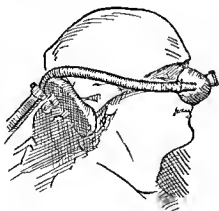


Fig 571—Nasal mask (McKesson). Gas passes in through the tubes under slight positive pressure, and passes out through the escape valve in the dome of the mask.

bation. A rubber Magill tube (Fig 573) may be inserted through the nose and passed between the cords either blindly or with the aid of a laryngoscope. In the second method the cords are exposed with a laryngoscope and the endotracheal tube is passed through the mouth and into the trachea under full vision. The tube may be of rubber, or of latex, or of coiled wire covered with Penrose tubing (Fig 574). In

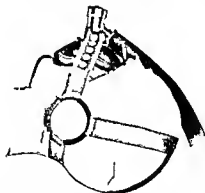


Fig 572—Ordinary surgical mask (McKesson) (Not suited to most otolaryngologic surgery.)

flatable rubber cuff surrounding the tube in the region of the larynx (Fig 577).

Any of the inhalation anesthetics may then be used.

Agents—Nitrous oxide and ethylene have little anesthetic power and produce little or no toxic effects. The liquid anesthetics are much more powerful and are correspondingly more toxic. All are inflammable and *explosive* except nitrous oxide and chloroform.

Nitrous oxide is so very weak an anesthetic that, if hypoxia is to be avoided, the patient's

reflex activity must usually be depressed by heavy preliminary medication or by simultaneous administration of ether, cyclopropane, or pentothal, or by avertin. Although nitrous

induce anesthesia before a switch is made to ether or to some other powerful anesthetic, and to serve as a vehicle for ether, and to supplement pentothal anesthesia

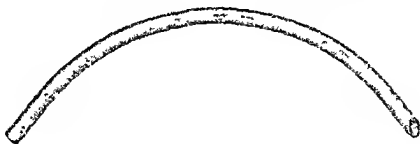


Fig. 573 —Rubber endotracheal tube (Magill). This may be passed through the mouth with the aid of a laryngo scope or through the nose blindly

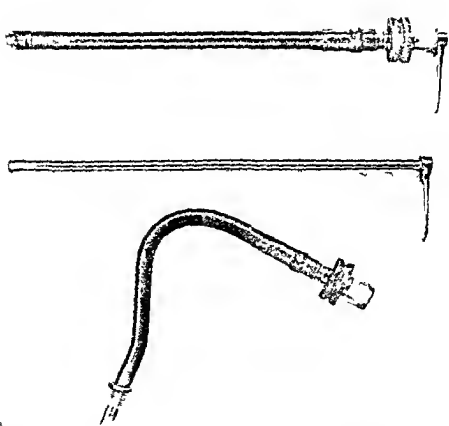


Fig. 574 —Flexible metal endotracheal tube (Flagg Woodbridge). Above, Stylet holds tube rigid for insertion with laryngoscope. Below, Stylet removed. Curved wire is inserted to show flexibility of the tube. Penrose tubing is rolled back at tip to show coiled wire construction

oxide itself is noninflammable, yet it supports combustion as does oxygen. Mixtures of nitrous oxide and ether are therefore inflammable and may be highly explosive. Nitrous oxide is non-toxic if hypoxia is avoided. It is chiefly useful to

Ethylene is only slightly more powerful. It is a very safe anesthetic, with a wide range of usefulness. Cyclopropane or ether may be added to it when greater anesthetic power is needed.

Cyclopropane is the only inhalation anesthetic to combine good power with low toxicity. It is, however, prone to cause cardiac arrhythmias, which in rare cases are fatal. It is an extremely

moderately toxic to many parts of the body, and is therefore contraindicated in the presence of hepatitis, nephritis, diabetes, marked debility, starvation from any cause, and acute respi-

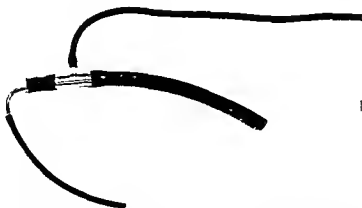


Fig. 575—Apparatus for semi-open endotracheal technique (Ayre) Magill endotracheal tube below. Tube for supply of ether vapor above. Large inhaling and exhaling tube between. (Lundy)

useful anesthetic when the closed system with carbon dioxide absorption may be used. It is contraindicated when epinephrine or other similar drugs are to be used. It may, however, be used with neosynephrin. It is also contraindi-

cated in otolaryngology. Ether and cyclopropane are the two most generally useful inhalation anesthetics in otolaryngology.

Vinyl ether is powerful and is rapid in action. It is much more pleasant to take than is ether,

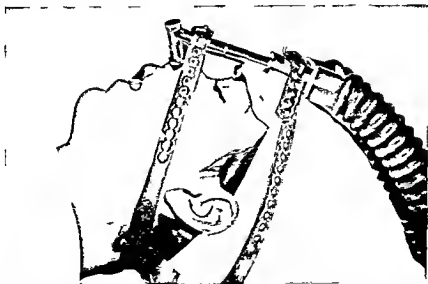


Fig. 576—Endotracheal tube connected to closed rebreathing system of gas machine, with absorption of carbon dioxide (Magill tube Adams adapter). Tube may be passed through either the nose or the mouth. (Lundy)

cated if the patient is digitalized or if cardiac rhythm is irregular.

Ether, because of its anesthetic potency and its cheapness, may be used by insufflation as well as by the other inhalation methods. It is

and emergence from anesthesia is more rapid. Its toxic effects on the liver limit its use to a total of one half hour in any ten day period and prohibit its use in the presence of hepatitis. Its chief uses are for inducing anesthesia before

ether is started, for brief operations such as myringotomy, and for painful dressings to be done in the patient's room

Ethyl chloride is probably not nearly so safe as vinyl ether. Although it is much more pleasant for the patient to take, yet it is recommended that it be replaced by vinyl ether.

Chloroform is too toxic to use unless a powerful noninflammable inhalation anesthetic is required.

Intravenous Anesthesia—Pentothal Sodium—The intravenous method presents the great advantage that the apparatus is out of the surgeon's way. There are, however, important drawbacks to the use of pentothal for otolar-

may be used to keep the glottis open. It is thus evident that the use of an endotracheal tube greatly widens the usefulness of pentothal.

Since the drug is broken down in the body and is excreted by the kidneys, it should be avoided in the presence of nephritis and probably of hepatitis also, and its use for complete anesthesia should be limited to one hour. It may be used for longer periods if anesthesia is obtained by a local anesthetic, and the pentothal is used merely to produce sleep.

Curare—With the patient already under a general anesthetic, curare may be given intravenously to produce deep relaxation for an otherwise difficult bronchoscopy. There are a

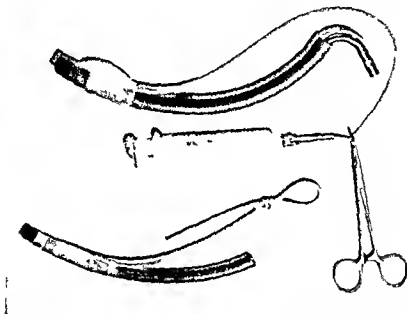


Fig. 577.—Inflatable cuff on Magill endotracheal tube. Below, Deflated. Above, Inflated by syringe and kept inflated by hemostatic clamp. (From Gillespie: *Endotracheal Anaesthesia*. The University of Wisconsin Press.)

ryngologic surgery. It causes considerable depression of the respiratory center, and sufficient relaxation frequently to bring about obstruction to breathing. For these reasons it may often be well to insure the patency of the respiratory tract by the use of an endotracheal tube, and to supply oxygen. Furthermore it increases rather than depresses the reflex activity of the larynx, so that in the presence of sufficient stimulus there is apt to be marked and dangerous spasm of the larynx and of the bronchial tree. It is therefore to be avoided in operations which may cause irritation of the pharynx or larynx unless the reflex is previously blocked by local anesthesia or unless an endotracheal tube

few important drawbacks to its use. The margin between production of relaxation and of respiratory paralysis is so narrow that one should always be ready to give artificial respiration with oxygen. Various lots of curare differ in composition and action, and its clinical use is still in the experimental stage.

Local and Regional Anesthesia—Agents—*Procaine* ("novocaine") is commonly used by injection methods in 0.5 to 2 per cent solution in physiologic saline. It is ineffective when applied topically. *Meperidine*, used in the same strengths, appears to take hold more promptly, to give slightly deeper anesthesia, to last a little longer, and to be no more toxic than procaine.

It produces fairly good anesthesia when applied topically in solutions of from 1 to 10 per cent in saline. Solutions keep indefinitely, and are self-sterilizing to a degree. *Pontocaine* in 2 per cent solution produces good anesthesia when applied topically. Clinically its toxicity does not seem to be greater than that of cocaine, and may be less. However, as in the case of cocaine, deaths have resulted from its use topically. It has been used, but not widely, for injection anesthesia in 0.1 per cent solution. *Cocaine* is still probably the most commonly used anesthetic for topical application. It is to be hoped that other newer topical anesthetics will prove to be less dangerous. It is too toxic for injection into the tissues. *Benzocaine* promises to be the best and the least toxic topical anesthetic now available. It is only slightly soluble in water, but is used in propylene glycol, in 10 per cent solution. When applied to the surface it does not penetrate so deeply as does cocaine. It does not cause local ischemia and shrinking.

Toxic Effects—The toxic effects of local anesthetics fall into two groups:

1. *Depression of circulation and of respiration*, as shown by pallor, sweating, fall of blood pressure, and shallowness of breathing. There may be nausea and vomiting. In injection anesthesia this is usually prevented by adding small amounts of epinephrine to the local anesthetic solution (0.2 to 0.4 cc. of 1:1000 solution for each gram of procaine or metycaine). Treatment consists in the use of the shock position, oxygen inhalation, epinephrine intramuscularly, and fluids intravenously.

2. *Irritability of the central nervous system*, culminating rapidly in *convulsions*. Death may result. Every patient who is to have local anesthesia should receive a barbiturate beforehand to prevent this type of toxicity. Treatment consists in the immediate administration of sufficient barbiturate intravenously to control the convulsions, and of oxygen inhalation. An endotracheal tube may help in getting oxygen into the lungs.

It appears likely that most cases of so called idiosyncrasy to local anesthetics are rather cases of accidental intravenous injection, or of rapid absorption of the drug into the blood stream, or of substitution of one drug for another by mistake, such as cocaine for procaine.

If excessive amounts of epinephrine are used, apprehension, tachycardia, hypertension, and pallor will result. These symptoms usually sub-

side in fifteen minutes without treatment, and the administration of the local anesthetic without epinephrine may then be continued. It is possible that the intravenous administration of pentothal might be helpful when symptoms develop. Epinephrine should be omitted in the presence of hyperthyroidism, and probably also if cyclopropane or chloroform is to be used.

Technic for Operations on the Neck—In the absence of local sepsis, regional anesthesia is suitable for most of the operations commonly performed on the neck, including resection of esophageal diverticulum, laryngectomy, and block dissection of cervical lymph nodes. It produces satisfactory analgesia for the usual maneuvers of cutting, hemostasis, and sewing, but occasionally traction on the esophagus or trachea produces a troublesome sense of nausea or of choking. For this reason manipulation should be gentle and opiates should be given rather liberally.

PARAVERTEBRAL BLOCK—With the patient supine, the operator stands facing the vertex of the patient's head. When the head has been turned sharply to the right, the left transverse processes of the lower cervical vertebrae may be felt as a vaguely defined ridge extending from above the middle of the clavicle toward the mastoid process. The three needles for paravertebral injection are to be placed in a line which passes along this ridge and through the mastoid process. The wheel for the uppermost or superior needle is raised in this line just below the mastoid process (Fig. 578). The wheel for the inferior needle is made where this same line is crossed by a perpendicular dropped from the upper border of the thyroid cartilage (Fig. 578, insert). The third wheel is placed half-way between these two.

An 8-cm. needle is then passed through the superior wheel in a plane perpendicular to the sagittal plane of the head. The needle is pointed slightly inferiorly or caudad, so that it will impinge on the transverse process of the second cervical vertebra near its tip. If it were not pointed inferiorly but were inserted in a direction perpendicular to the skin, it might pass between two transverse processes and puncture either the dura or the vertebral artery, which runs through the foramina of the transverse processes. While the needle is being inserted with the right hand, the fingers of the left hand should constantly palpate the ridge of transverse processes in the inferior part of the neck,

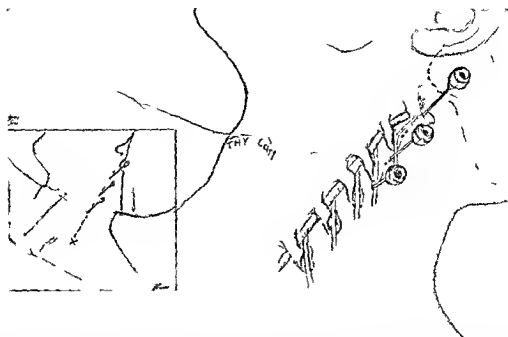


Fig 578 —Paravertebral cervical nerve block. Broken lines show ridge of transverse processes to mastoid process, and perpendicular dropped from upper border of thyroid cartilage. Note proximity of vertebral artery to needles (Author's article in *Lahey Clinic issue of Surgical Clinics of North America*, 19)

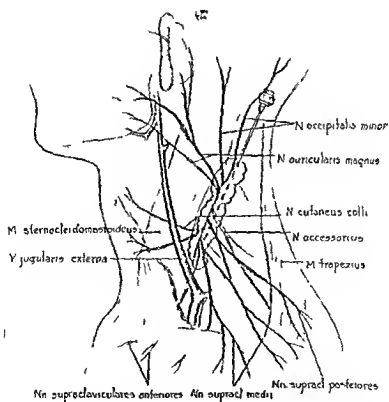


Fig 579 —Poststernomastoid block of superficial branches of cervical plexus (Author's article in *Lahey Clinic issue of Surgical Clinics of North America*, 19)

for it is the superior part of this ridge, which is difficult to palpate, that the needle must reach. If the operator is in doubt as to the proper direction for the needle, he should first direct it

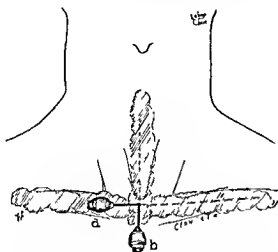


Fig 580—*a*, Line of infiltration between clavicle and skin to supplement other injections for operations that approach the clavicle *b*, Infiltration in anterior midline of neck, to supplement unilateral injections for operations that approach the midline (Author's article in *Lahey Clinic issue of Surgical Clinics of North America*, 19)

too far posteriorly, and successively withdraw and insert it in more anterior directions until the point strikes the bony ridge, because there are few structures posterior to the transverse processes that could be injured by the exploring needle. When the point strikes bone at a depth sufficiently close to the skin to indicate that it is on or near the tip of the transverse process, the syringe is connected to it and aspiration is made in order to make sure that the point is not in a blood vessel or in the dural sac. Six cubic centimeters of 1 per cent solution of metycaine or of procaine is injected. The injection should be made slowly, and aspiration should be repeatedly performed.

A second needle is similarly placed through the middle wheal. It may conveniently be 5 cm long, because in this location the transverse process is nearer the skin. The same amount of solution is injected through it. This process is repeated with a 5-cm needle through the inferior wheal.

The next injection is along the *sternomastoid* muscle (Fig 579). An 8-cm needle, attached to the syringe, is inserted through whichever wheal lies nearest the posterior border of this muscle. The point pierces the platysma and comes to lie at the depth of the body of the sternomastoid

muscle. Ten cubic centimeters of solution is injected along the middle half of its posterior border. The needle passes deep to the external jugular vein. This injection fortifies the anesthesia of the superficial branches of the cervical plexus: the small occipital, the great auricular, the superficial cervical, and the anterior, middle, and posterior supraclavicular nerves.

FIELD BLOCKS—Anesthesia is completed by one of the following field blocks, according to the location of the proposed incision. For this purpose a 0.5 per cent solution is used.

If the incision is to extend near the clavicle, as for excision of esophageal diverticulum, a wheal is raised just below the sternal notch, an 8-cm needle is inserted through the wheal, and a line of 0.5 per cent solution is deposited between the clavicle and skin (Fig 580, needle *a*). This serves to block off any branches of upper intercostal nerves that might overlap with the sensory supply from the cervical plexus.

If the operation is to involve the upper part of the neck, the overlapping fibers of the fifth cranial nerve are blocked. This is done by a line of subcutaneous infiltration from the second or middle wheal to the angle of the mandible, and thence along the border of the mandible to the point of the chin (Fig 581, needle *a*). If the operation is to involve also the region of the submental and submaxillary

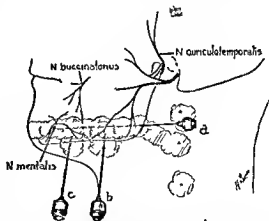


Fig 581—Supplementary injections for operations which approach the mandible *a*, Infiltration along border of mandible *b*, *c*, Infiltration in floor of mouth. (Author's article in *Lahey Clinic issue of Surgical Clinics of North America*, 19)

glands, the floor of the mouth should be infiltrated. A needle is inserted through the skin one-third of the way from the angle of the mandible to the point of the chin, and directed

toward the eye (Fig 581, needle b) It makes contact with the lower border of the mandible, is then withdrawn and is reinserted just medial to the mandible, and 0.5 to 1.0 cm deeper than before. Five cubic centimeters of 0.5 per cent solution is injected. A similar injection is made at a point two thirds of the way from the angle to the point of the chin (Fig 581, needle c).

If the operation is to involve both sides of the neck, the entire injection or any part of it may be made on the other side also without fear of trouble from paresis of the diaphragm.

If the operation is to be limited to one side and is to approach the midline at any point, a line of infiltration should be made along the midline in order to anesthetize fibers that overlap from the other side (Fig 580, needle b).

For operations which may involve dislocation of the thyroid gland, the paravertebral and poststernomastoid blocks are done as described above, and an additional injection is made just anterior to the midpoint of the anterior border of the sternomastoid muscle. The needle is inserted perpendicularly to the skin until its point reaches the level of the deep surface of that muscle. With frequent aspiration, 5 cc of 0.5 per cent solution is injected. As an alternative or supplement to this injection, the surgeon may infiltrate the region of the superior pole of the thyroid after he has exposed it.

For operations on the larynx, the paravertebral and poststernomastoid injections are made, and in addition the superior laryngeal nerves are blocked in the following manner. The greater cornu of the hyoid bone is made prominent by pressure on the cornu of the opposite side, a wheal is raised 1 cm inferior and 2 cm anterior to the extremity of the cornu, and through it a needle is passed, parallel to the hyoid bone and directed posteriorly between it and the upper border of the thyroid cartilage. The point of the needle must not pass posterior to the posterior extremity of the hyoid bone. The patient is instructed to raise his hand (rather than to speak) when there is any sensation referred to the ear. If paresthesia is obtained, 2 cc of 2 per cent solution is immediately injected. If none is obtained, injection is made in the needle's tract while it is being withdrawn. If coughing or stridor occurs, the injection should be interrupted immediately.

For laryngectomy, the surgeon should in addition infiltrate 0.5 per cent solution pos-

terior to the larynx after the dissection has approached this region.

Choice of Anesthesia.—The reader is referred also to the first paragraph of this chapter (p 778).

Local anesthesia is usually preferable for most operations on the nose, for tonsillectomy in adults, for laryngofissure, for tracheostomy, for most superficial operations, and for procedures involving the use of the bronchoscope or esophagoscope. For tonsillectomy under general anesthesia for small children the pharyngeal insufflation of ether appears most suitable. In larger children and in adults it is preferable to use gas with a rubber endotracheal tube through either the mouth or the nose. Inhalation of matter foreign to the trachea may then be prevented by maintaining slight positive pressure and by leaving the tube in place after the close of the operation until the laryngeal reflex returns.

For myringotomy, brief anesthesia may be produced with vinyl ether, or with one of the gases or with pentothal. For operations on the middle ear general anesthesia with the use of the endotracheal tube is usually preferable. The anesthetic agent may be any of the gases, or pentothal.

For laryngectomy a variety of methods is available. Regional anesthesia may be used, as has been described. Regional or local infiltration anesthesia may be combined with narcosis produced by a moderate dose of avertin or by a light plane of pentothal anesthesia. The avertin is not subject to satisfactory control, and the temptation with pentothal is to give a dangerously large total amount. It appears to be more satisfactory and safe to give one of the gases with an endotracheal tube. Before the trachea is transected, the tube is withdrawn, and the trachea is pierced with a hypodermic needle and a few drops of a topical anesthetic are injected into its lumen. As soon as the trachea is cut across a sterilized endotracheal tube is put into the lower segment of the trachea and is connected to the gas machine. Gauze is then packed lightly between the tube and the trachea, and slight positive pressure of the gas is maintained. These measures prevent entry of blood into the trachea and escape of an unduly large amount of gas.

Whenever any form of general anesthesia is used under conditions which may lead to con-

tioned obstruction to breathing, an endotracheal tube should be used

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OTORHINOLARYNGOLOGIC SURGERY FOR PATIENTS WITH PULMONARY TUBERCULOSIS

It is a well known fact that patients with pulmonary tuberculosis do not tolerate even minor surgery well. First of all the employment of an anesthetic is hazardous. Very few textbooks on anesthesia give more than brief mention of the importance of the anesthetic for the tuberculous patient. Ether as a general anesthetic is actually dangerous, as it aggravates the pulmonary disease. Nitrous oxide, ethyl chloride, somnoform, ethylene, vinyl ether, cyclopropane, and similar agents are safer than ether (Chloroform is almost never used today as an anesthetic agent, except in small amounts in obstetric work.) Using oxygen with the anesthetic lessens the danger, but it does not make employment of inhalation anesthesia safe. Local anesthesia is safest for the patient with pulmonary tuberculosis.

Aside from the problem of anesthesia there are other points to be considered when surgery

is contemplated for the tuberculous patient. In such patients the fever is increased following surgical procedures and often remains higher for a long time after recovery from the operation. Loss of appetite and loss of weight are almost certain to follow even minor surgery. Most patients with pulmonary tuberculosis have found it difficult to gain weight. Ordinarily they must overeat in order to maintain weight. To gain weight a good appetite is very necessary. Secondary anemia commonly accompanies pulmonary tuberculosis and this condition is aggravated by even minor surgery. Moreover, surgical shock is more likely to occur in the presence of anemia.

It is impossible, in a short treatise such as this, to mention and discuss all of the major and minor otorhinolaryngologic operations which may be required by patients with pulmonary tuberculosis. However, sufficient procedures will be discussed to show the surgical principles and hazards involved which usually do not require consideration in the nontuberculous. Dr. Wm J. Mayo once said, "a good surgeon is one who knows when not to perform an operation." His statement is doubly true for the tuberculous. "Fools rush in where angels fear to tread" has no place in surgery and surely not for the tuberculous patient.

Acute mastoiditis is fortunately a rare disease in tuberculous patients. Every effort should be made to avoid surgery. With the aid of the sulfa drugs and penicillin this may be accomplished. If an operation becomes imperative it should be performed under local anesthesia with the aid of morphine and the barbiturates. Radical mastoid surgery should be postponed until the pulmonary tuberculosis has been arrested or cured for one or two years except when intracranial complications threaten or are actually present. Mastoid wounds in the tuberculous patient present many problems in delayed healing. Granulation tissue forms slowly and is quite pale, or it doesn't form at all.

Sinus surgery should be restricted to simple drainage, such as a simple antrotomy of the maxillary sinus or the removal of the anterior one-third of the middle turbinate with or without the uncapping of a few ethmoid cells for drainage of a frontal sinus. Uncapping the posterior ethmoid cells or enlarging the natural ostium of the sphenoid sinus may be required in rare cases. Local anesthesia should be used. Submucous resection of the nasal septum can

usually be postponed until the pulmonary disease has been arrested for one year

Tonsillectomy and even *adenoidectomy* present a different problem. The dangers of a tonsillectomy in the tuberculous patient were emphasized by Mullin and me in the discussion of the paper by Newhart, Cohen, and Van-Winkle¹ read before the American Laryngological Association in 1934. Many patients with pulmonary tuberculosis have minute mouse nibbled superficial tuberculous ulcers of the pharyngeal mucous membranes. These are difficult to diagnose, because in the early stages there may be no symptoms and the lesions are difficult to see. Spraying the pharynx with a 1 or 2 per cent aqueous solution of fluorescein, made slightly alkaline by the addition of sodium bicarbonate, will stain the ulcers a pale green color. If these ulcers are present a tonsillectomy, even under local anesthesia, will cause them to enlarge, dysphagia or even odynophagia may result. An adenoidectomy is safer than a tonsillectomy, as the ulcers have a predilection for the hypopharynx.

Cauterization of the tuberculous larynx, under local anesthesia, is one minor surgical procedure well tolerated in well selected cases. High fever, rapid ulceration of the larynx, necrosis of cartilage, very active pulmonary tuberculosis, and marked secondary anemia are some of the contraindications to cauterization. I have tried to present these in my book on laryngeal tuberculosis.²

Peroral endoscopy for diagnosis and treatment has been well tolerated in well-selected cases. The very high degree of scientific attainment given this surgical procedure by Chevalier Jackson³ has made this a very safe method.

Tracheotomy in the tuberculous, if not put off until too late, should be a reasonably safe procedure under local anesthesia. The relief from the obstruction to breathing and the general improvement which follows often justify this oldest of all operations. The procedure is a humane one as advocated by Jackson.³ Myerson⁴ has presented the indications for tracheotomy in patients with laryngopulmonary tuberculosis most thoroughly. He has shown that in properly selected cases the operation is beneficial.

Abscesses may have to be drained wherever they form. An acute otitis media may require a myringotomy under local anesthesia, an abscess of the septum may have to be incised, an

acutely infected maxillary sinus may require a needle puncture and washing, an abscess of the epiglottis may likewise require incision, retropharyngeal, peritonsillar, and similar abscesses may require opening. These are often minor emergencies and should not be classed with operations of election, such as a tonsillectomy.

Dentists may ask about the advisability of extracting one or several *infected teeth* for a patient with pulmonary tuberculosis. The sulfa drugs, administered for a day or two preceding the extraction, have made the danger of post-operative infection much less. The extraction is a reasonably safe minor procedure which can be well done under local anesthesia even in the tuberculous patient.

Operations for malignant disease, such as carcinoma and sarcoma, present a very serious complication of pulmonary tuberculosis. These tumors produce a secondary anemia and loss of weight just as tuberculosis does, so that the patient usually cannot stand the shock of surgery. Roentgen ray and radium therapy often have to be used without previous surgery. Each case must, of course, be judged by its merit and what seems to be the best treatment for the particular patient. Knowledge and experience produce the best judgment.

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equation of the physician. Some otorhinolaryngologists see their private patients only in a special or general hospital. Others maintain a private hospital of their own. Some have operating and recovery rooms in connection with their consulting rooms, with equipment and personnel necessary for performing all except major operations on seriously ill patients. A few are dependent on their own individual efforts with the occasional help of a member of the family. Much good work is done by general practitioners who have had unusual opportunities for acquiring special knowledge in otorhinolaryngology and skill in its application. The immense progress that has been made in this field, and in medical science, has greatly changed conditions in recent years. In the early days of the specialty the equipment was relatively small and inexpensive. Today there is a large proportion of surgical work required for treatment of diseases of the regions concerned, and there is a large amount of diagnostic and therapeutic work that is dependent upon facilities for routine laboratory examination, including microscopic, histologic, bacteriologic, hematologic, biologic, roentgenologic, and other studies. These developments require either a large outlay for equipment or a plan by which the supplementary facilities are to be provided. This applies chiefly to diagnosis. If to this be added the equipment for operations, for the application of therapy, and for such laboratory work as the gaging of blood concentration of arsenic, bismuth and the sulfonamides for example, the outlay is more than doubled. Moreover, distracting problems of personnel, organization, and administration become involved. Group practice, or seeing private patients in a well equipped and fully staffed hospital with an administrative organization of its own takes an enormous load of details and executive care off the shoulders of the otorhinolaryngologist, leaving him free for his clinical work. Each individual must decide for himself how much of this supplementary work he wishes to do, or is required by circumstances to do.

Perhaps the majority of physicians specializing in this department of medicine have consulting rooms with screened corners or cubicles equipped for examining patients, larger space for functional tests, and at least a small room equipped for doing some of the laboratory work. Patients requiring complicated studies and probably operation are put in a hospital

for completion of diagnostic work as well as for operation, if this should prove to be indicated.

For such a plan the consulting room or secretary's room should have a case record file, the simpler it is the more likely will it afford the essential data when wanted for future reference. Card files are simplest but require another file for supplementary records or reports as well as for correspondence.

An examining cubicle should not be less than about 2 m (about 6 or 7 feet) in depth and width. It should contain a good examining light, a chair for the patient, a stool for the examiner, an instrument cabinet, a small instrument sterilizer, and air under positive and negative pressures. The examining instruments and the stock of drugs used in examination and local treatment should be such as the otorhinolaryngologist has found most useful in his clinical work or postgraduate studies. More than one cubicle will save much time for a busy practitioner. The wall of the cubicle should be tinted green and the general light in the cubicle should be subdued. In addition to the examining cubicle there should be a room that can be made absolutely dark for best results in transillumination. A room larger than a cubicle and having walls, floor, and ceiling lined with sound proof material is necessary for functional tests of hearing and equilibrium. As this room should be windowless it might well be arranged also for transillumination. If the otorhinolaryngologist is to do any laboratory work, such as microscopic, histologic, biologic, hematologic and bacteriologic examinations, tests for blood concentrations in chemotherapy, and the like, another room equipped as a laboratory would be essential and probably before long would require the services of a technician.

Complete Examination.—Fundamental to all treatment of the nose, throat, and ear is local examination. It is unjustifiable, except in emergency, to treat any one of these regions, or in fact give treatment for any complaint of the head or neck, without thorough and complete examination of all of them. There is abundant evidence to show that, except in emergency, no patient should be treated for any illness, in any region without such an examination. Indeed, there is much to be said in support of periodical complete examinations, say annually, in absence of symptoms, as is now generally accepted regarding the teeth. In presence of a single

symptom referable to any region there can be no question as to the advisability of examination of all regions. Documentary evidence of this is afforded by going over the records of a series of cases of a chronic disease, cancer for example. It is deplorable to note the number of patients coming in with incurably late lesions that could have been discovered in the curable stage had a complete instead of a regional examination been made earlier. Of course the psychologic phase of the matter must be considered. Many patients resent complete examinations, some because of expense especially for such procedures as roentgen ray examinations, others are critical of the ray examination of the chest when the complaint is of a discharging ear.

Many years ago the writer made it a rule that every patient must have roentgen-ray, serologic, and general medical examinations, and to expedite the study of out of town patients these studies were started routinely by secretaries. This arrangement met with many protests, chiefly on three bases. Patients of limited means who wanted to pay their way saw their small fund dwindle before the larynx was examined, other patients demanded laryngeal examination to determine whether or not the various studies were necessary. In a third class of patients the claim was made that all accessory studies had been made at home, but they brought no reports or roentgen ray films, and often the claimed examinations had been made months previously. A plan then adopted proved satisfactory in all the years since. A tactful secretary first took a record starting with the name and address followed by the chief complaint and as many facts concerning the family history, previous illness, and present illness as could be tactfully obtained. Usually it was complete, it all ways rendered possible a beginning with the examination of the region of chief complaint. To illustrate the psychology involved it may be said that if a patient comes to the otorhinolaryngologist because of earache it would be untactful to begin with examination of the larynx, notwithstanding the possibility that the pain in the ear might be due to a laryngeal lesion. Conversely, if he comes complaining of cough starting with a "tickling" in the larynx it would be best to start with examination of the larynx, notwithstanding the possibility that impacted cerumen might be causative. The important point is that though a complete otorhinolar-

ynologic examination should be made in every case, satisfaction and cooperation on the part of the patient depend upon recognition of the psychologic phase that the patient is concentrated on the region of chief complaint. Parenthetically, it might be added that throughout all subsequent contact with that patient that chief complaint should be always kept in mind.

By complete otorhinolaryngologic examination we mean examination of the nasal cavities, accessory sinuses, mouth (including teeth and tongue), fauces, pharynx, nasopharynx, larynx, ear, and neck. The methods of making these regional examinations are given in previous pages. Then the eyes should be examined for pupillary reaction, muscular imbalance, clarity of media, and, if obtainable without a mydriatic, the general appearance of the fundus. *Complete general medical examination should follow.* A specimen of blood is taken for examination and for serologic test as soon as possible in order that reports may be soon available, the same applies to roentgen ray studies, many decisions cannot be made until these two classes of reports are received. The next step is a thorough physical examination, including heart, lungs, abdomen, skin, reflexes, endocrine system, and so on. During all of these examinations a constant watch is maintained for evidence, however slight, of psychopathic irregularities. With all of these data in hand the otorhinolaryngologist or the bronchoesophagologist is prepared to determine what further studies, such as biopsy, need be done to complete the diagnosis and the therapeutic plan. Obviously this plan requires modification in certain cases. The patient may have been referred by a well qualified practitioner who has made, or has had made, some or all of the examinations and has sent reports and good, recent, adequate films. Urgency or an emergency, such as impending asphyxia, dehydration, starvation, hemorrhage, shock, or unconsciousness, may require postponement of complete examination. As a general rule, however, a specialist, highly trained in a narrow field, risks doing an injustice to his patient unless he has for consideration all of the data yielded by a complete medical examination. The best of all ways is, of course, to have a conference with conferees including always a general medical man, or an internist, or pediatrician. In most cases the pathologist and roentgenologist are invaluable participants. Unfortunately, with the

exception of hospital staffs and clinical groups, such conferences are not practicable, and the practitioner must rely upon his knowledge, skill, experience, and available literature. Such a task to most physicians would be a pleasure if time were abundant, but again unfortunately, it is difficult for the practitioner overburdened with clinical work to carry out complete general examination. It may be added that it is marvelous what good work is done under the difficult conditions mentioned.

The foregoing hears chiefly on diagnosis. The complete general examination has an equally important bearing on treatment. In nonoperative cases in the field of otolaryngology the treatment of chronic diseases, and of frequently repeated attacks of acute diseases, will often be disappointing and insufficient unless all general disorders are recognized and properly treated. Apart from allergic conditions and such specific diseases as tuberculosis and syphilis, it must be remembered that local treatment of chronic mucosal diseases will be inadequate if such general conditions as avitaminosis and endocrinopathy are overlooked. *Avitaminosis* though not fully understood can always be efficiently treated. Too much effort need not be made to select the particular vitamin that is deficient. A perfectly balanced diet will go a long way toward helping in the treatment of all chronic otorhinolaryngologic diseases, and as an excess of vitamins in the form of pharmaceutical preparations can do no harm it is well to supplement the diet with a test of the effect of complex preparations offered by the leading manufacturers of medicinal drugs and chemicals who maintain biologic laboratories. Attention to avitaminosis is also of utmost importance preliminary to operation. Prompt healing and good postoperative condition are promoted by such attention, and occasionally the indications for the operation will be found to have disappeared with the correction of the deficiency.

When the general medical examination reveals *endocrine imbalance* no treatment should be undertaken, except in emergency, without proper management of the endocrinopathy. It is true there remain many unsolved problems in the large fields of endocrinology and its bearing on otorhinolaryngologic disorders, but treatment of these disorders will be greatly helped by endocrinotherapy. Not only is there interrelation among the ductless glands, but involvement of the vegetative nervous system may in-

fluence many organs. Before operation, correction of minor degrees of hypo-activity, such as tetany, or of hyperactivity, such as hyperthyroidism, is an essential part of good surgery. In some cases such preliminary treatment may even modify or remove the indications for operation. Major degrees of diseases of the ductless glands may contraindicate a contemplated operation, such as one for betterment of a chronic ailment, or any operation not urgently and immediately necessary. Even if they do not contraindicate the operation the surgeon should know beforehand of their existence and should have on record in his files any suspicion of such conditions as cretinism, myxedema, tetany, Addison's disease (suprarenal hypofunction), Banti's disease (or other form of splenic disorder or associated hepatic cirrhosis), hypophysial disorders (especially the infantilism and adiposity of hypopituitarism), and the involvement of the anterior pituitary lobe in sphenoidal or nasopharyngeal disease, or vice versa. Well-developed cases of these and similar disorders have symptoms that are obvious, but in other cases the underlying disorder is discoverable only by the alert clinician, and may be readily overlooked by the surgically-occupied otorhinolaryngologist.

Matters Relevant to Operations—Consent is necessary before even a minor operation. In case of an emergency such as copious hemorrhage or impending asphyxia the operator has a good defense if he can afterward prove the urgency calling for what he did. It is well to know that consent if given may have to be proven, therefore it should be obtained in the presence of one or more *disinterested persons* or, best of all, in writing, and it should cover any operation deemed necessary by the surgeon. If done routinely on a blank there is usually no difficulty in this. If a patient cannot trust the surgeon's judgment he should not entrust his life by consent to any operation. Even when in writing a consent may be invalid if signed by a minor, an unqualified, an unauthorized, or a deranged person. In case of estranged parents, the consent of either parent is sufficient, in case the other parent protests, the operator should further protect himself by obtaining medical opinions that would be available afterward to prove the necessity for the operation, though, ordinarily, necessity, in absence of an extreme emergency, does not imply consent. One naturally wishes to avoid the unfavorable effect of

depressing a patient before operation, yet it will not do to take out a man's larynx, for example, without telling him beforehand just what his postoperative condition will be.

Most persons dread operations on themselves, and most parents dread operation on their children, yet in some psychotic and psychopathic states a recital of symptoms calling for operation may be based upon knowledge acquired in various ways and used in an effort to bring about an unnecessary operation even though it be a painful one. Somewhat similar conditions may arise from hypochondriasis with which every physician is familiar, but it should also be borne in mind that hypochondriac fixation is sometimes an initial symptom in schizophrenia (dementia precox). The operator's records should always show the basis for the advice for operative treatment. None of the mental conditions ranging from morbid introspection to dementia can be benefited by operation, all require psychiatric treatment. On the other hand a patient with violent dementia may be remarkably submissive to treatment of an acute otitis media, for example.

Psychotic and Psychoneurotic Patients—There are other phases of the disorders mentioned in the foregoing paragraph. The surgically-occupied practitioner is prone to drift into purely physical habits of thought. The avoidance of pitfalls as well as the best interests of his patients require that he be always alert to the possibility of a patient having a psychoneurosis or a tendency thereto. Dementia and imbecility are readily recognized, but the more obscure and some of the early stages require for their detection elementary knowledge of the fundamentals of psychiatry. Every otolaryngologist should have such knowledge, and especially so as many psychoses are first and often chiefly manifested by complaints referred to the special senses—taste, smell, hearing, sometimes vision—or to the functions of equilibrium and swallowing. Headaches and various hyperesthesias and paresthesias due solely to psychoses may simulate diseases of the nose, throat, or ear.

A few of the symptoms of mental derangement will be mentioned. The onset of dementia may be sudden, violent, and unmistakable. Often it is so insidious as not to be beyond what would be normal limits for another individual. In such cases early changes in behavior are to be determined only by comparison with

preceding behavior of the same person. Loss of mental power may at first be no more than would be natural for old age, such as failure of memory for recent events, or slight inability or confusion in mental effort. These often are to be noted by an alert observer in the course of an examination that includes record of events and symptoms, tests of hearing, localization and description of sensations, and the like. More significant symptoms may be noted. There may be excitement, in which there is an abnormal output of mental energy, or there may be depression, with slow actions, difficult thinking, misery, and unhappiness. Between these two extremes is apathy, manifested by lack of interest in anything, although, if stimulus be applied, mental processes are found not lacking in a degree of clarity. Hallucinations, in which the patient sees things or hears voices that do not exist, differ from delusions, which are beliefs, often in personal grandeur or persecution. Obsession is a borderline symptom of, seemingly, no significance if taken alone, as, for example, cancerphobia or a hobby. Alterations of speech may be symptoms of an incipient mental instability, or of organic disease such as is so often seen by the otolaryngologist. Amnesia in a righthanded patient with disease of the left ear might seem to be an amnesic aphasia calling for opening of the middle fossa, yet it might be recognized by a psychiatrist as significant of an early stage of mental derangement. Loquacity may be found, if closely followed, to be a roundabout way of avoiding nouns that cannot be remembered (anomia), or it may be the beginning of the incoherent speech or pedantic phraseology of a psychosis. Loss of speech may be an obsession or a delusion denoting a psychotic or psychoneurotic development, or, it may be a fraudulent claim with a purpose, a phase of malingering, or a psychic trauma, or it may be due to discoverable organic disease within the field of otolaryngology.

The recognition of and differential diagnosis among the innumerable phases of the psychoses, psychoneurosis, psychopathic states, conversion and anxiety factors, emotional instability, and various borderline conditions involve complex problems for the psychiatrist, but it is well for the otolaryngologist to avoid pitfalls by having always in mind the possibility of such disorders, and by making it a rule in his decision to attach greatest weight to the objective

evidences of disease elicited in his examinations. The methods of obtaining these evidences, and guidance in their interpretation, will be found on preceding pages in connection with the various diseases.

Litigation.—Few things are more annoying to the busy clinician than attendance at court as either a witness or a litigant. Of course, under all circumstances it is well to get legal advice, but a few fundamentals may be stated here. A physician may appear in court as a common, professional, or expert witness. A common witness is one who has had, or is supposed to have had, knowledge of facts to which professional confidence does not apply. Any such person can be hauled into court and compelled to testify under penalty of imprisonment for contempt. There may be a pittance as a witness fee and under some circumstances a reimbursement for traveling expenses may be allowed, but ordinarily attendance as a common witness is a total loss. A physician who has been consulted by a patient cannot, ordinarily, be compelled to divulge any confidential medical information concerning the patient given him by the patient or the patient's relatives. There are certain circumstances, chiefly criminal, in which this does not apply. A patient may waive his right of professional confidence, but the waiver should be in writing unless it is of record in court. Expert witnesses are usually called, not by the court, but by the litigants, plaintiff, or defendant, in civil cases, appearance of such experts in court is optional the first time, but after testifying recall may be obligatory. Depositions may be taken out of court but under arrangement affording opportunity for cross examination. Expert witness fees should be but are not fixed by the court. The remuneration, if any, comes from the litigant calling in the expert witness. It may be added that, unless careful in their answers, opposed experts may make themselves and medical science appear ridiculous to the laity. A distinction should be made between facts and opinions, also between controversial and generally accepted opinions. A witness is expected to answer questions not to ask them. If he does not understand a question he should say so and avoid asking a question in order to obtain clarification. Some attorneys in cross-questioning try to browbeat a witness. The best way to meet this is to preserve unruffled poise with a sincere effort to give judge and jury the information that will

help them in arriving at just conclusions. Any attempt at repartee is usually inadvisable, however great the temptation may be. The otorhinolaryngologist or the bronchoesophagologist may be called upon for testimony as an expert, or as a common witness if the rights of professional confidence are waived, in cases involving claims for indemnity or compensation. Such claims may be against insurance companies, transportation corporations, or employers, and are usually based on loss of time, pain, and suffering or permanent, partial, or total disability. Questions of facts, dates, and the like should be available from the physician's records, but it is often a difficult problem to determine the extent of injury and to exclude previously-existing disability or disease. These matters and the extent and duration of future disability are in the nature of expert testimony, and the physician should be prepared for technical cross examination. The limitations of medical science, as well as simple justice, call for conservatism in expression of expert opinions on the witness stand.

A physician may become involved innocently or unexpectedly as a *defendant* in litigation, and often such involvement could have been avoided by a little knowledge of the subject. In reporting cases in medical journals care is necessary to avoid giving any names, or other information that could be used to identify the patient, and photographs should be rendered unrecognizable. These precautions apply whether the case involves implications of things about which the patient might be proud or those concerning which he might be embarrassed. A practitioner, general or special, becomes an accessory after the fact if he fails to report to authorities in case of operation changing appearances, of the nose for example, if the patient should turn out to be wanted by the police. A similar possibility may be involved in case of treatment of a fresh wound. A practitioner, general or special, notwithstanding he is licensed by the state, is under no legal obligation to take a patient for treatment, and he would probably not be liable for any first aid treatment given in an emergency, but once he renders medical service in a case he becomes legally liable, particularly in two ways. First, he risks liability if he fails to give the patient the benefit of skill and judgment such as is afforded by practitioners in the same line of work in the same community. Unless he so represents himself the specialist in a remote

village is not required to afford the same skill or equipment as the unique specialist in a great medical center (though of course, he may have such skill and equipment) The general practitioner is not required to have the skill and experience of the specialist, and, legally, he becomes liable if he undertakes a case requiring such qualifications, except as a matter of first aid or other emergency There is no implication of guarantee of cure in any case, the burden of proof of a specific guarantee would rest on the claimant

The other important way in which any practitioner, general or special, may become liable is by *negligence* in any one of many forms A claim may be based not only on neglect of attendance but on neglect to use proper skill and judgment at the proper time The surgeon is not liable for an accident during operation, unless the accident was probably caused by negligence on the part of himself or his assistants or employees, or of the nurse or other employees of the hospital who are working under the surgeon's direction, but when an accident occurs the operator becomes liable if he does not promptly take what measures may be necessary for the patient's best interests For example, in three different cases a Watson Williams rasp broke during a nasal operation and the fragment of rasp was inspirated into a bronchus.^{1 2} In each case the patient had no ground for action because the rhinologist promptly told the patient of the accident, arranged for a roentgen

ray examination which located the fragment, and referred the patient to a presumably competent bronchoscopist.^{1 2} One of the three patients referred to claimed that bronchiectasis resulted from the presence of the rasp Our records of bronchoscopic and roentgen ray examinations showed conclusively that the bronchiectatic tissue changes were of many-years' standing and could not possibly have resulted from the recent three-weeks' sojourn of the foreign body To support a claim of negligence the claimant must be free of contributory negligence For example, if a patient should refuse or neglect to follow advice to have a roentgen-ray examination his contributory negligence would stand against his claim for indemnity, even if there had been negligence on the part of the defendant physician Another principle of law is that one cannot contract against his own negligence For example, it is no defense against a claim of negligence for a physician to prove that he warned the patient of the possibility of being too busy to care for the patient

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